6. BASELINE RISK ASSESSMENT

The Waste Area Group (WAG) 1 baseline risk assessment (BRA) is divided into two evaluations: a human health risk assessment (see Sections 6.1 through 6.6) and an ecological risk assessment (ERA) (see Section 7). The human health risk assessment approach used in the BRA is based on the *EPAs Risk Assessment Guidance for Superfund (RAGS)*, (EPA 1989a), the *INEL Track 2 Guidance Document* (DOE-ID 1994), and the *INEL Cumulative Risk Assessment Guidance Protocol* [Lockheed Martin Idaho Technologies Company (LMITCO) 1995]. Similarly, the ERA approach used in this assessment is based on the *INEL Screening Level Ecological Risk Assessment Guidance Document* (VanHorn, Hampton, and Morris 1995).

Preliminary evaluations of both human health and ecological risks at WAG 1 have been completed as part of the *OU 1-10 RI/FS Work Plan* (Lewis et al. 1996). The WAG 1 screening and data gap analysis (SDGA) report [Attachment I of the Remedial Investigation/Feasibility Study (RI/FS) Work Plan] was developed as a preliminary evaluation of WAG 1 human health risks, and the WAG 1 screening level ecological risk assessment (SLERA) (Attachment VIII of the RI/FS Work Plan) was developed as a preliminary evaluation of WAG 1 ecological risks. Most of the calculation methods used in this BRA were successfully implemented in the SDGA and SLERA.

This BRA is consistent with the Operable Unit (OU) 1-07B Record of Decision (ROD) because the BRA evaluates post-remedial action groundwater concentrations for the OU 1-07B contamination plume. As discussed in Sections 6.3 and 6.5, the BRA assumes that the OU 1-07B remedial action will be successful and that only residual groundwater contamination will be left in the aquifer after the remediation is complete.

A discussion of general comprehensive risk assessment methodologies is presented in the *INEL Cumulative Risk Assessment Guidance Protocol* (LMITCO 1995). As discussed in this document, the analysis methods used in Idaho National Engineering and Environmental Laboratory (INEEL) comprehensive risk assessments are often different from the analysis methods used in *INEL Track 1 and Track 2 Risk Assessments* (DOE-ID 1994). In general, the differences between the two types of analyses are present because comprehensive risk assessments are meant to analyze risks produced by multiple release sites within a WAG, while Track 1 and Track 2 risk assessments are only meant to analyze risks from one release site at a time.

To satisfy the broader objective of INEEL comprehensive risk assessments, the *INEL Cumulative Risk Assessment Guidance Protocol* recommends analyzing risks produced through the air and groundwater exposure pathways in a "cumulative" manner. A cumulative analysis of these two exposure pathways involves calculating one WAG-wide risk number for each contaminant of potential concern (COPC) in each air and groundwater exposure route (e.g., inhalation of fugitive dust, ingestion of groundwater, etc.). Analyzing the air and groundwater pathways in a cumulative manner is necessary because contamination from all release sites within a WAG may affect air and groundwater exposure pathways at the WAG. Conversely, individual release sites within a WAG are typically isolated from one another with respect to the soil pathway exposure routes (e.g., ingestion of soil, ingestion of homegrown produce, etc.). As a result, the guidance protocol recommends analyzing soil pathway exposures on a release-site-specific, or "noncumulative," basis in INEEL comprehensive risk assessments.

The details of the "comprehensive" and "cumulative" aspects of the WAG 1 BRA are discussed more completely in the following sections. In general, the BRA is "comprehensive" because it evaluates

risks from all known and potential release sites within WAG 1, and it is "cumulative" because risks from multiple release sites are evaluated in the air and groundwater exposure pathways.

The term "risk" is used throughout this section in a generic sense. Generally the term is used to refer to the possibility of adverse health effects from either carcinogenic or noncarcinogenic contaminants, however, it is also used when only carcinogenic health effects are being discussed. The term "hazard quotient" (HQ) is used when only noncarcinogenic health effects are being discussed.

6.1 Baseline Risk Assessment Tasks

In general, the tasks associated with development of the WAG 1 human health and ERA are activities as follows:

- Data evaluation, including site and contaminant screening

- Exposure assessment

- Toxicity assessment

- Risk characterization

- Uncertainty analysis.

These tasks are described in the following subsections.

6.1.1 Perform Data Evaluation

All sampling data collected to date at WAG 1 release sites (see Section 4 for a discussion of the various WAG 1 sampling investigations) were evaluated to determine whether the data are appropriate and adequate for use in the BRA. This evaluation was conducted in accordance with *EPAs Guidance for Data Usability in Risk Assessment* (EPA 1992a). As part of this analysis, sampling data sets were assumed to have lognormal distributions in accordance with *EPAs Guidance on Calculating Concentration Terms* (EPA 1992b); however, statistical distributions for the data were not determined.

The data evaluation tasks that were completed as part of the BRA are as follows:

- Identification of release sites and co-located facilities that require further evaluation. Co-located facilities are defined as operating or shutdown facilities that have the potential for producing future releases of hazardous substances (see Section 6.6.5)

- Screen of release sites to identify sites that have the potential to produce adverse human health and ecological impacts (see Section 6.2.1 for a discussion of the site screening process)

- Review of available sampling data for the retained release sites. This review included a "process knowledge" evaluation designed to identify any contaminants that may have been released at a given site but not sampled for.

- Identification of contaminants detected at each retained release site and screened to identify COPCs (see Section 6.2.2 for a discussion of the contaminant screening process)

- Identification of potential exposure routes for each COPC

- Development of data set for use in the risk assessment.

The results of the data evaluation tasks are presented in Section 6.2.

6.1.2 Conduct Exposure Assessment

The process of exposure assessment quantifies all receptor intakes of COPCs for selected pathways. The assessment consists of estimating for COPCs the magnitude, frequency, duration, and exposure route to humans and ecological receptors. The following exposure assessment tasks were performed as part of the BRA process:

- Identification and characterization of exposed populations

- Identification of complete exposure pathways

- Estimation of contaminant concentrations at points of exposure

  - Soil pathway

  - Air pathway

  - Groundwater pathway

- Estimation of human intake rates

- Calculation of intake factors.

The conceptual site model (CSM) used to develop the BRA exposure assessment is presented in Figure 6-1, and the results of the exposure assessment tasks are presented in Section 6.3.

6.1.3 Conduct Toxicity Assessment

Toxicity assessment is the process of characterizing the relationship between the dose or intake of a substance and the incidence of an adverse effect in the exposed population. Toxicity assessments evaluate results from studies with laboratory animals, or from human epidemiological studies. These evaluations are used to extrapolate from high levels of exposure, where adverse effects are known to occur, to low levels of environmental exposures, where effects can only be predicted based on statistical probabilities. The results of these extrapolations are used to establish quantitative indicators of toxicity.

Health risks from all routes of exposure are characterized by combining the chemical intake information with numerical indicators of toxicity. These health-protective toxicity criteria are obtained through Environmental Protection Agency (EPA)-developed reference doses (RfDs) or slope factors (SFs). The information used as part of the BRA toxicity assessment is presented in Section 6.4.
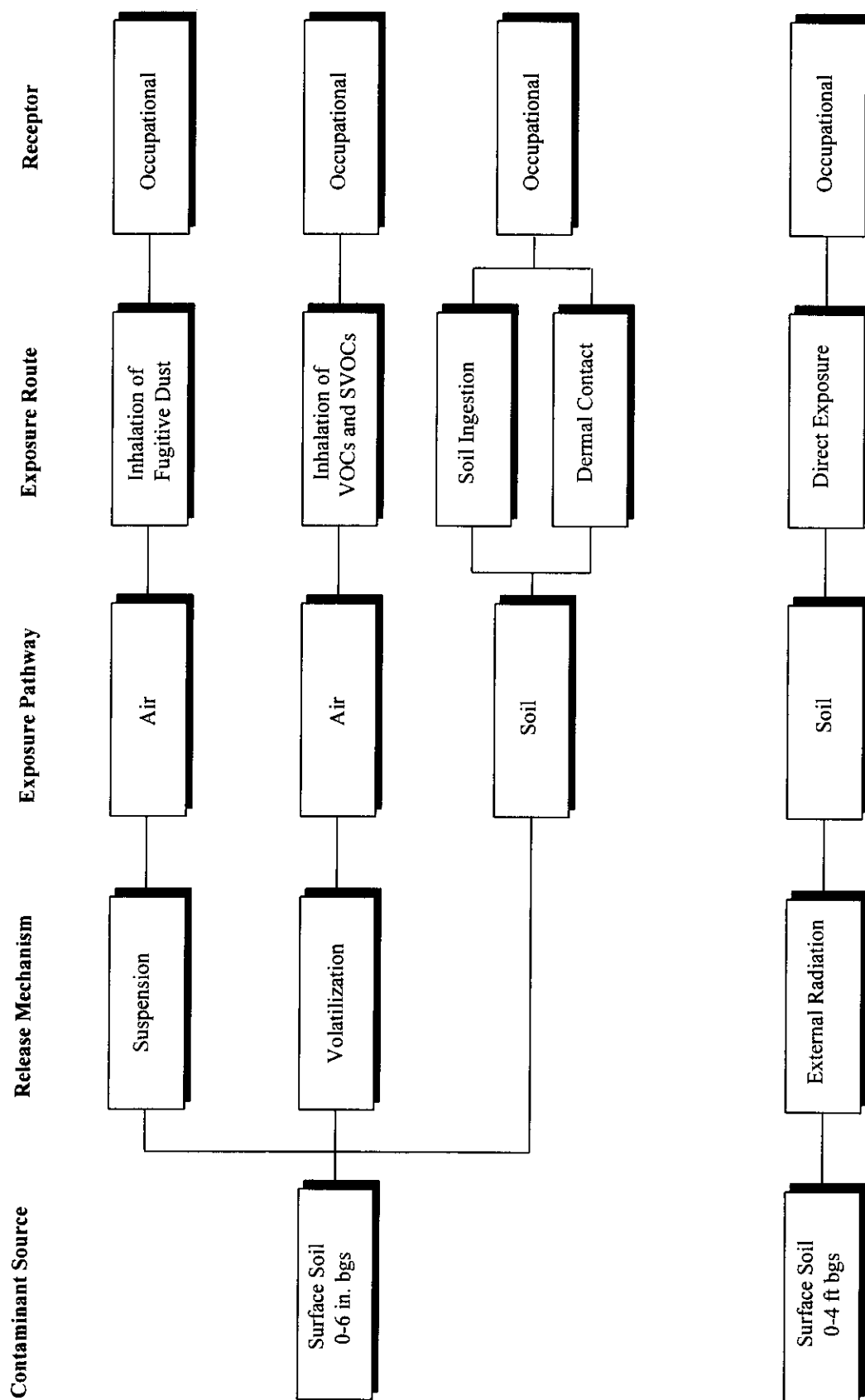
Figure 6-1. Occupational exposure scenario conceptual site model.
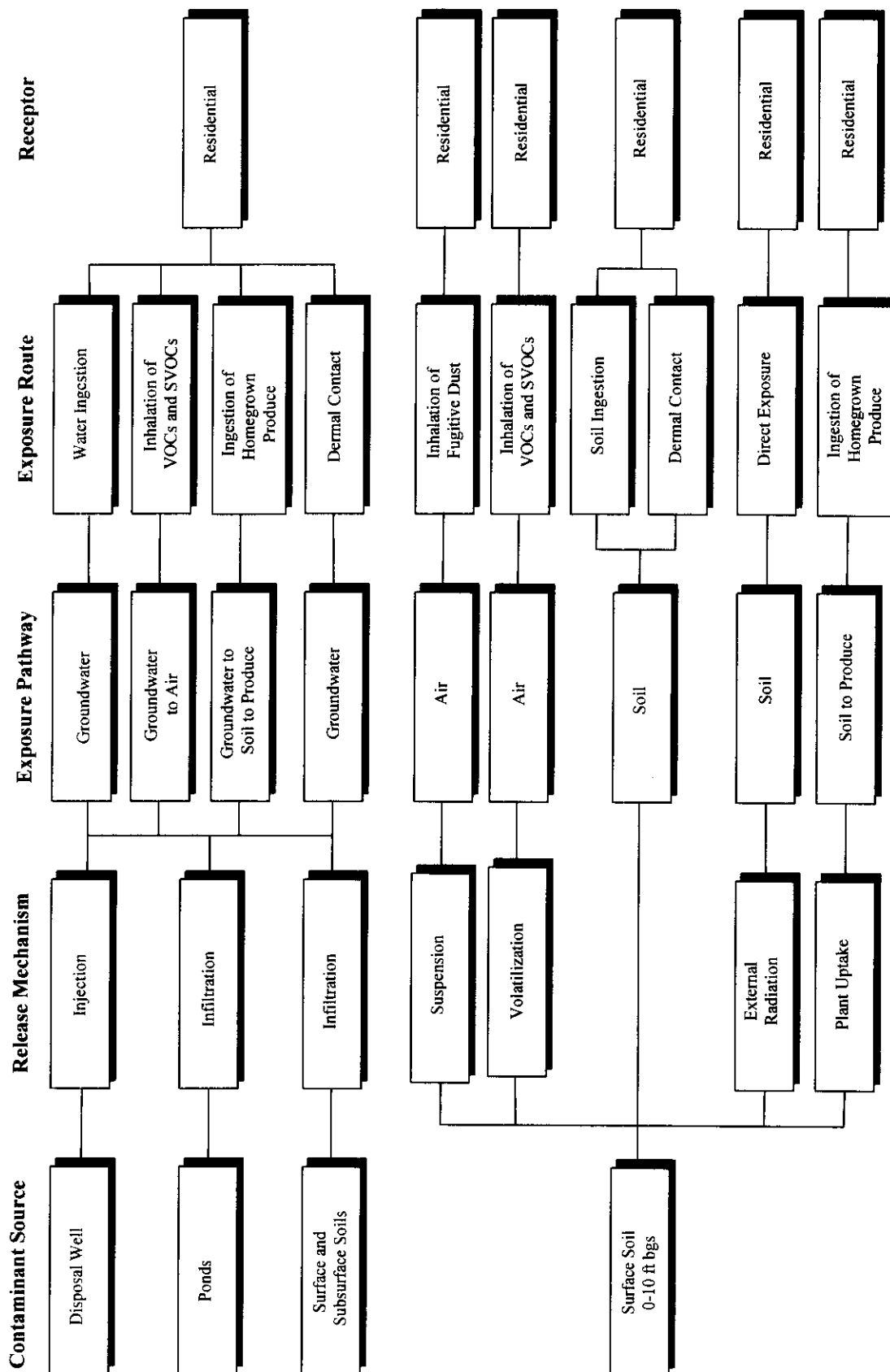
Figure 6-1. (continued). Residential exposure scenario conceptual site model.

6.1.4 Risk Characterization

Risk characterization involves combining the results of the toxicity and exposure assessments to provide a numerical estimate of health risk. This estimate is either a comparison of exposure levels with appropriate toxicity criteria, or an estimate of the lifetime cancer risk associated with a particular intake. Risk characterization also considers the nature and weight of evidence supporting the risk estimate, as well as the magnitude of uncertainty surrounding the estimate. The results of the BRA risk characterization process, including risk estimates for each of the retained release sites, are presented in Section 6.5.

6.2 Site and Contaminant Screening

This section presents the site and contaminant screening methodologies used in the WAG 1 BRA. These screening methodologies are used to help focus the BRA by identifying release sites and contaminants that do not contribute to the comprehensive human health or ecological risk at WAG 1. The screening methodologies are designed to be conservative so that only sites and contaminants that clearly do not pose any threat of producing adverse human health or ecological effects are identified by the methodologies.

For the remainder of this report, sites and contaminants that pass the screening processes will be referred to as "retained" sites and contaminants; all retained sites and contaminants are further evaluated in Sections 6.3 through 6.6. Likewise, sites and contaminants that fail the screening processes will be referred to as "eliminated" sites and contaminants. All eliminated sites and contaminants will receive no further evaluation in the BRA.

Only historic release sites that have been identified at WAG 1 are considered in the OU 1-10 site and contaminant screening processes. Test Area North (TAN) facilities that may pose a threat of producing future contamination releases are not included in these screening evaluations, because all future releases are assumed to have the potential for causing adverse human health and ecological effects. Potential future releases from TAN facilities are discussed as part of the WAG 1 co-located facilities evaluation presented in Section 6.6.

Figure 6-2 presents a graphical description of the site and contaminant screening methodologies discussed in the following sections.

6.2.1 Site Screening Methodology

Table 6-1 presents a list of WAG 1 release sites. All of the sites listed in this table are considered in the site screening process.

The following steps are used to screen release sites:

1.   The contaminant sampling information for all WAG 1 sites is compiled.

2.   Sites that have not been evaluated by previous risk assessments (i.e., new sites) are identified.

3.   Federal Facility Agreement/Consent Order (FFA/CO) no action sites are eliminated. These are sites that are identified as requiring no further action in the FFA/CO.
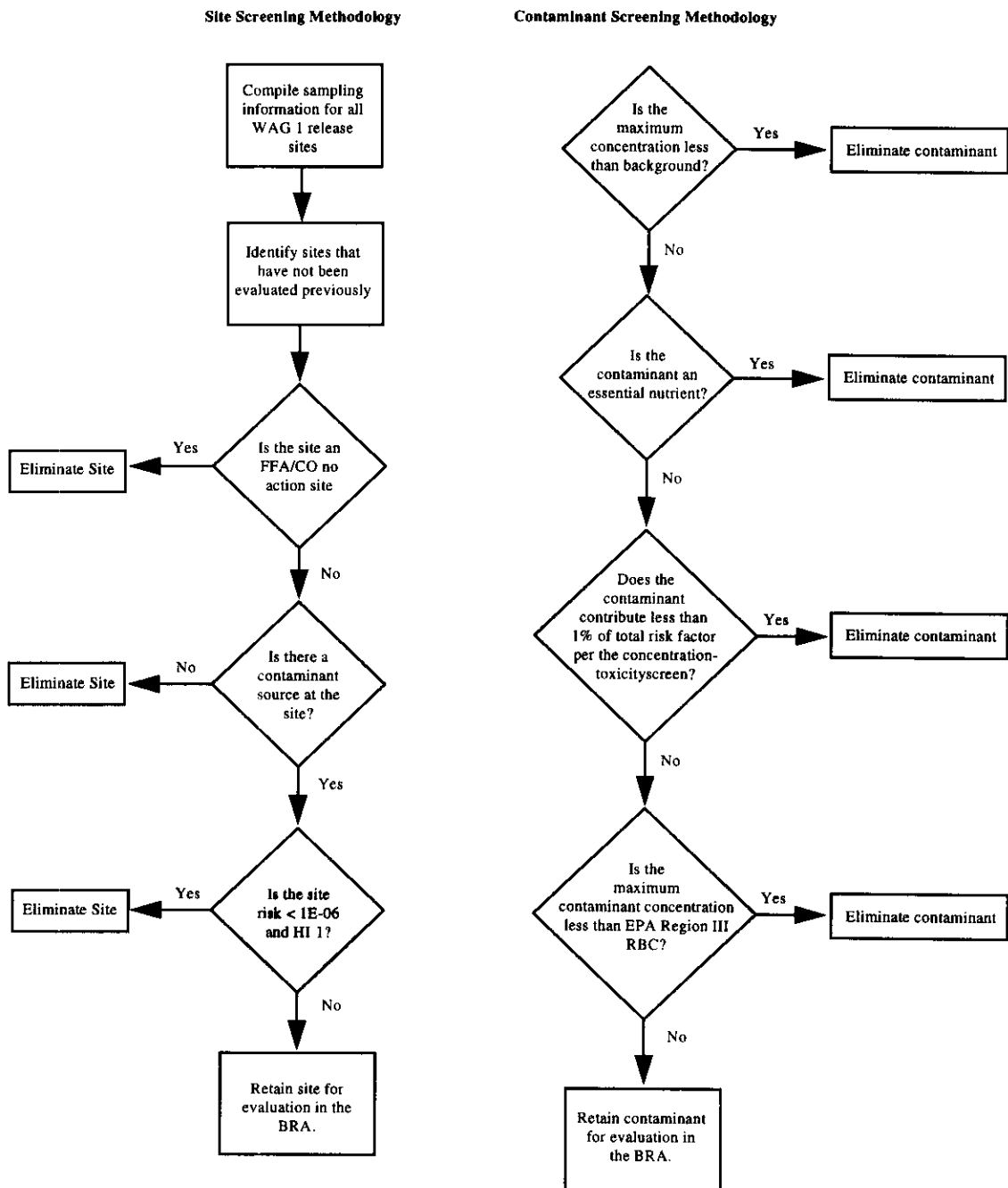
Figure 6-2. Site and contaminant screening methodologies.

Table 6-1. Summary of WAG 1 release sites and COPCs considered in the BRA.

OU	Subunit	Site Description	COPCs	Contaminated Medium or Media
1-01	TSF-11	Technical Support Facility (TSF) Three Clarifier Pits East of TAN-604	Am-241	Subsurface soil
1-03	TSF-03	TSF Burn Pit	2-methylnaphthalene, phenanthrene, lead	Subsurface soil
1-03	WRRTF-01	Water Reactor Research Test Facility (WRRTF) Burn Pits I, II, III, and IV	2-hexanone, 2-methylnaphthalene, phenanthrene, chromium, lead, Co-60, Pu-239/-240	Subsurface soil
1-03	TSF-38	TSF Bottle Site	None	Not applicable
1-04	TSF-29	TSF Acid Pond	Am-241, Cm-242, Cm-243/244, Cs-137, Np-237, U-235	Surface soil and shallow subsurface soil
1-05	IET-04	Initial Engine Test (IET) Stack Rubble Site	Fixed radionuclide contamination	Not applicable
1-05	TSF-06	Area 1	Co-60, Cs-137, Th-232, U-238	Surface soil and subsurface soil
1-05	TSF-06	Area 3	Am-241	Surface soil
1-05	TSF-06	Area 5	Co-60, Cs-137	Surface soil and shallow subsurface soil
1-05	TSF-06	Area 7	Co-60, Cs-134, Cs-137	Surface soil and shallow subsurface soil
1-05	TSF-06	Area 8	Co-60, Cs-134, Cs-137, Sr-90	Surface soil and subsurface soil
1-05	TSF-06	Area 9	Cs-137	Surface soil and shallow subsurface soil
1-05	TSF-06	Area 10	None	Not applicable
1-05	TSF-06	Area 11	Co-60, Cs-137, Eu-154	Surface soil and shallow subsurface soil

Table 6-1. (continued).

OU	Subunit	Site Description	COPCs	Contaminated Medium or Media
1-05	TSF-06	Area B	Co-60, Cs-137	Surface soil and shallow subsurface soil
1-05	TSF-09/-18	TSF Intermediate-Level (Radioactive) Waste Disposal System and Contaminated Tank Southeast of Tank V-3	Co-60, Cs-134, Cs-137, Eu-154	Surface soil and subsurface soil
1-05	TSF-10	TSF Drainage Pond	Manganese, Cs-137	Surface soil and subsurface soil
1-05	TSF-21	TSF IET Valve Pit	Co-60, Cs-137	Subsurface soil
1-05	TSF-26	TSF PM/2A Tanks	Co-60, Cs-137	Surface soil and subsurface soil
1-05	WRRTF-04	WRRTF Radioactive Liquid Waste Tank	None	Not applicable
1-06	TSF-07	TSF Disposal Pond	Aroclor-1260, benzo(a)pyrene, n-propylbenzene, phenanthrene, propionitrile, tetrahydrofuran, arsenic, barium, mercury, thallium, sulfide, Am-241, Co-60, Cs-134, Cs-137, H-3, Ra-226, Th-234	Surface soil and subsurface soil
1-06	TSF-08	TSF Heat Transfer Reactor Experimental (HTRE) Mercury Spill Area Site 13B	Mercury, Co-60, Cs-137	Subsurface soil
1-07A/B	TSF-05/-23	TSF Injection Well and Groundwater Contamination Beneath TSF	Tetrachloroethene, trichloroethene, cis-1,2-dichloroethene, trans-1,2-dichloroethene, Sr-90	Groundwater
1-08	TSF-22	TSF Railroad Turntable	Co-60, Cs-137	Surface soil and shallow subsurface soil

Table 6-1. (continued).

OU	Subunit	Site Description	COPCs	Contaminated Medium or Media
1-08	WRRTF-05	WRRTF Injection Well	Acenaphthene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, sec-butylbenzene, tert-butylbenzene, di-n-octylphthalate, dichlorodifluoromethane, isopropylbenzene, 2-methylnaphthalene, n-propylbenzene, naphthalene, 1,2-trimethylbenzene, 1,3,5-trimethylbenzene	Groundwater
1-08	WRRTF-13	WRRTF Fuel Leak	2-methylnaphthalene, phthalate, phenanthrene, total petroleum hydrocarbon (TPH)	Subsurface soil
1-09	TSF-36	TSF TAN-603 French Drain	Benzo(a)pyrene, TPH-diesel	Subsurface soil
1-09	TSF-37	TSF Contaminated Well Water Spill	H-3, Sr-90	Shall subsurface and subsurface soil
1-10	TSF-27	TSF Paint Shop Ditch	None	Not applicable
1-10	Loss-of-Fluid Test Facility (LOFT)-12	LOFT North Transformer Yard polychlorinated biphenyl (PCB) Spill and Soil Site	Aroclor-1260	Shallow subsurface soil

4. Eliminate sites for which a contaminant source does not exist. These are sites that have either never contained any contamination, or have had all contamination removed.

5. Eliminate sites for which risk was determined to be insignificant by previous risk evaluation activities (e.g., Track 1, Track 2, or other investigations). Risk and hazard index (HI) levels of 1E–06 and 1.0, respectively, are used for this screening step because fewer than 10 release sites are eliminated by this step.

6. Sites containing known contaminants are retained for further evaluation against the contaminant screening criteria.

6.2.1.1 *Step 1.* The contaminant sampling information for all WAG 1 sites is compiled. In the FFA/CO, WAG 1 is divided into 10 OUs, and these OUs are further divided into individual release sites. Figure 6-3 shows the location of the WAG 1 release sites identified in the FFA/CO, as well as the locations of release sites that have been identified since the FFA/CO was finalized [e.g., Water Reactor Research Test Facility (WRRTF)-13]. Release site descriptions are presented in Section 4, and a summary of the site descriptions is presented in Table B-1.

6.2.1.2 *Step 2.* Sites that have not been evaluated by previous risk assessments are identified. All sites that have not been evaluated by previous risk assessments (for example the Track 1 or Track 2 investigations), (DOE-ID 1994) are identified. In general, only sites that are not listed in the FFA/CO are identified by this screening step. These sites are not subject to Step 5 of the site screening process.

6.2.1.3 *Steps 3 through 5.* No action, no-source, and low-risk sites are eliminated. Table B-2 lists the justification for elimination (by step number) of individual release sites. Sites that are designated as no action in the FFA/CO, and as a result were not assigned to an OU, are eliminated by Step 3. Sites for which sampling has not detected a source, or where remedial action has removed all sources, are eliminated in Step 4. Finally, Step 5 eliminates all sites that have been shown to have risks less than 1E-06 and HIs less than 1.0 by Track 1 or Track 2 investigations. Screening criteria of 1E–06 and 1.0 are used because these levels are the minimum acceptable human health risk and HQ values cited in the National Contingency Plan (NCP) (see Section 6.5). Fewer than 10 release sites are screened by Step 5.
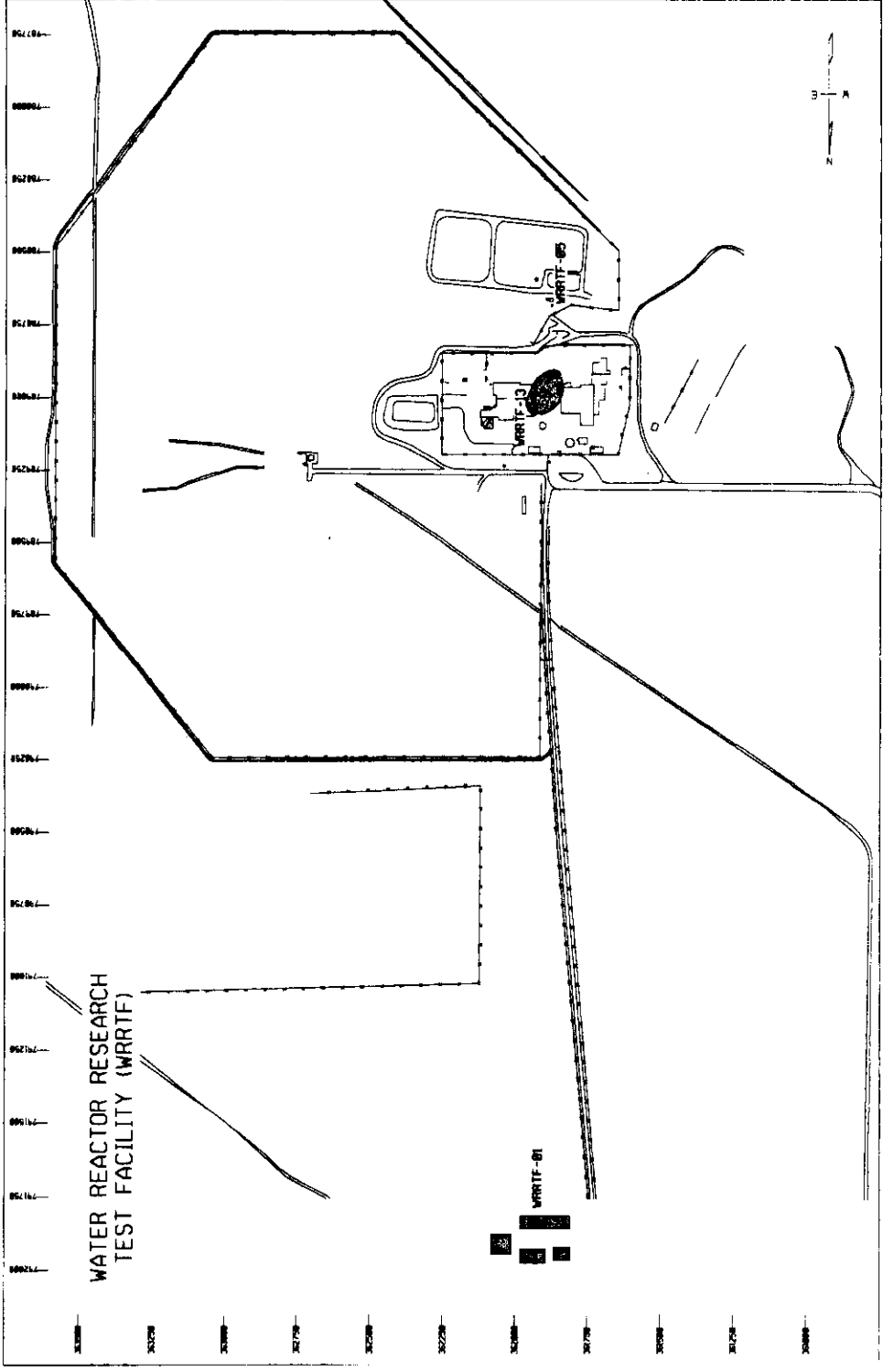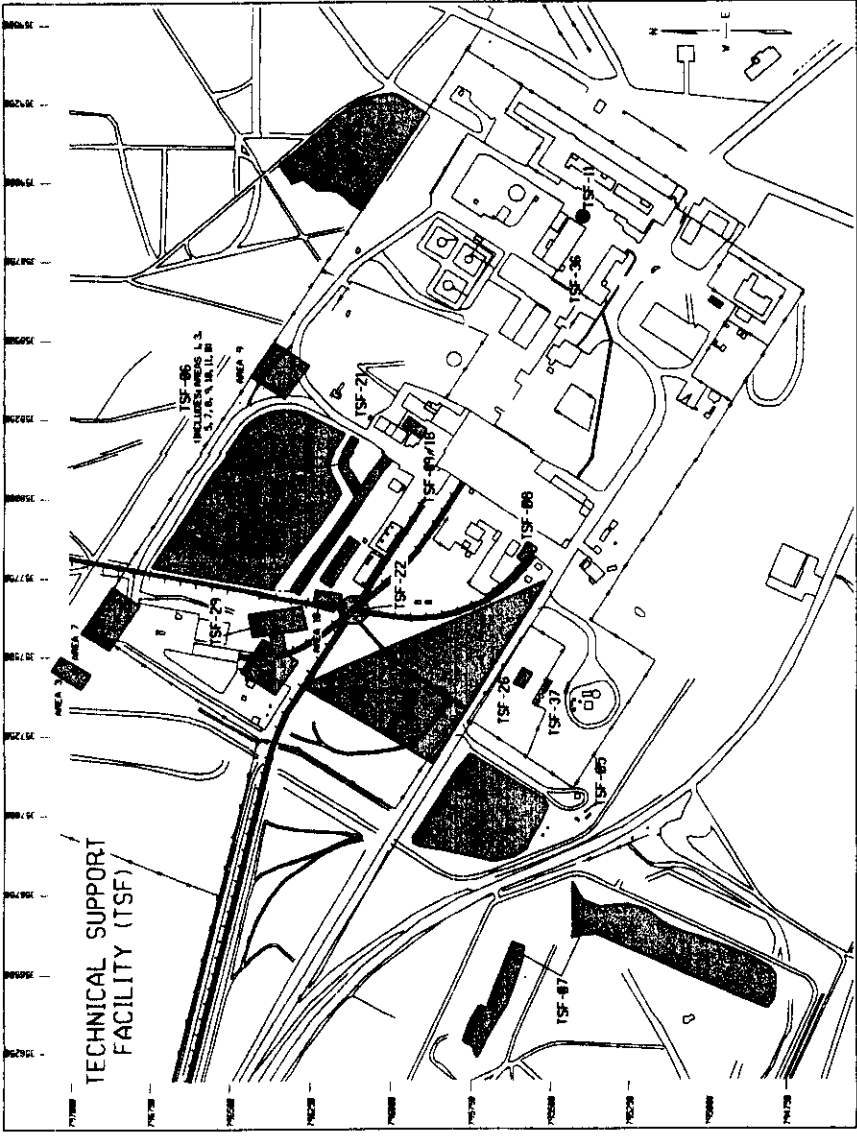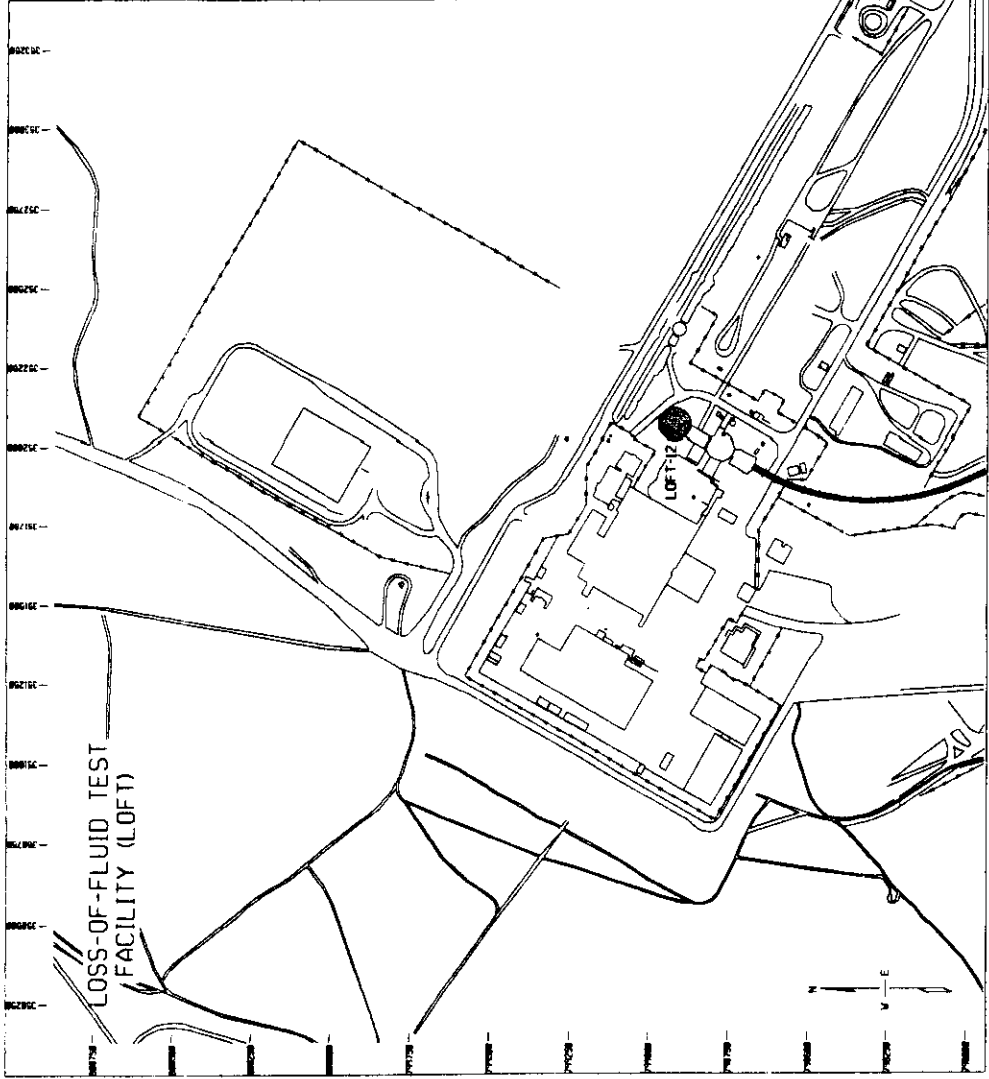
6.2.1.4 *Step 6.* Sites containing known contaminants are retained for further evaluation against the contaminant screening criteria. All sites that are not eliminated in Steps 3 through 5 of this process are retained for further evaluation. Table B-2 presents a list of all retained sites.

6.2.2 Contaminant Screening Methodology

The contaminant screening method depicted in Figure 6-2 involves compiling all sampling data for each retained site. The sources of sampling results used in the contaminant screening are various Track 1, Track 2, and other investigation reports, the WAG 1 SLERA (Attachment VIII of the RI/FS Work Plan), and the Integrated Data Environmental Management System (IDEMS) database. The IDEMS database manages INEEL sampling data, and ensures that the analysis data, methods, and data validation qualifiers for all organic, inorganic, and radiological data are consistent.

The following steps are used to screen contaminants. Each screening step is applied to each contaminant that has been detected at each retained site. As a result of the screening process, individual contaminants may be eliminated at one retained site, but retained at other sites.

TECHNICAL SUPPORT
FACILITY (TSF)

LOSS-OF-FLUID TEST
FACILITY (LOFT)

WATER REACTOR RESEARCH
TEST FACILITY (WRRTF)

SITE NAME	AREA (FT²)	VOLUME (FT³)
TSF-03	240,000	2,400,000
TSF-05	7,700	3,850
TSF-06, AREA 1	1,500	4,500
TSF-06, AREA 3	30,000	120,000
TSF-06, AREA 5	13,625	136,250
TSF-06, AREA 7		
TSF-06, AREA 8 (includes pre-1958 pond portion of TSF-29)	2,348	5850
TSF-06, AREA 9	4,500	11,250
TSF-06, AREA 10	160,312	480,780
TSF-06, AREA 11	185,480	1,354,480
TSF-06, AREA 8	810	6,498
TSF-07	4,800	190,080
TSF-09/10	28,260	451,200
TSF-11	40	120
TSF-21	2,491	18,007.5
TSF-22	6,362	15,905
TSF-26	7,800	332,500
TSF-29	245,500	68,000
TSF-36	78.5	235.5
TSF-37	78.5	314
LOFT-12	920	4,600
WRRTF-01	27,126	563,209

LEGEND:
Site Location
Fence
Stream or drainage

The OU 1-10 BRA contaminant screening steps are as follows:

1. All contaminants with maximum concentrations that are less than or equal to INEEL background concentrations are eliminated from further evaluation. Background concentrations were taken from *the INEL Background Guidance Document* (Rood, Harris, and White 1995). If a specific background concentration is not available, then the contaminant is retained and other screening criteria are considered.

2. Five inorganic contaminants that are routinely analyzed for but which are not associated with toxicity to humans under normal circumstances (i.e., calcium, iron, magnesium, potassium, and sodium) (EPA 1991) are eliminated. This screening step is only applied when the measured concentration of one of these contaminants is less than ten times the contaminant's background concentration.

3. Contaminants that are identified as having relatively low concentration and toxicity values through application of a concentration-toxicity screen are eliminated. As discussed in *EPA Risk Assessment Guidance* (EPA 1989a), the screen is applied by calculating a carcinogenic risk factor or a noncarcinogenic risk factor, for every nonradionuclide contaminant at each retained site. The factors for each type of contaminant are then combined, and any contaminant with a factor that is less than 1% of its category's compiled total is eliminated from further consideration. This screening step is not applied to radionuclide contaminants.

4. Contaminants that have maximum detected concentrations that are less than the most limiting risk-based concentration (RBC) values listed in an *EPA RBC Evaluation* (EPA 1995). The values shown in the RBC evaluation are the concentrations that will produce a calculated lifetime cancer risk of 1E-06, or a HQ of 1. The risk-based screening method is applied by comparing the maximum detected soil concentration for a given contaminant at a given release site against the most restrictive concentration for the contaminant shown in the RBC evaluation.

5. Contaminants that are not eliminated by the above four steps are retained for further evaluation.

The following equation is used to calculate concentration-toxicity screen risk factors (EPA 1989a):

$$R_{ij} = C_{ij} \times T_{ij} \tag{6-1}$$

where

R_{ij} = risk factor for contaminant i in medium j.

C_{ij} = concentration of contaminant i in medium j.

T_{ij} = toxicity value for contaminant i in medium j [i.e., either SF, or RfD^{-1} (1/RfD)].

There is a possibility that a contaminant may be present at a retained site without being detected by any site sampling investigations. These unidentified contaminants would not be included in the contaminant screening evaluation. The possibility of important contaminants escaping identification is considered to be small because most site sampling investigations were designed to detect all contaminants

that may have been released at a site, and a review of the processes that generated the contamination at each retained site was included as part of the BRA data evaluation process described in Section 6.1.1.

Tables B-3 through B-29 present the results of the contaminant screening evaluation. The tables show the maximum concentration of each contaminant found at each retained site, the background concentrations of contaminants, and whether a given contaminant is eliminated by the screening process. If a given contaminant is eliminated, the final column of each table gives the number of the contaminant screening step that caused the elimination. Measurement uncertainties associated with observed radionuclide concentrations have been included in these tables when the uncertainty levels are known.

An aspect of the BRA that tends to exaggerate risk results is the evaluation of contaminants with background concentrations that produce calculated risks in excess of 1E–06. One example of this type of contaminant is arsenic. Arsenic is commonly detected in INEEL soils at concentrations that are slightly higher than accepted background concentrations, however, the contaminant is not associated with known waste producing processes at WAG 1, and it has a high toxicity value. For these reasons, arsenic was eliminated from the BRA for five sites [Technical Support Facility (TSF)-9/18, TSF-10, TSF-26, TSF-27, and TSF-36)].

One site evaluated in the BRA (TSF-07) contains a maximum detected arsenic concentration that is significantly greater than background (49.2 mg/kg). This site was contaminated by wastewaters that were released into a disposal pond, and there is a possibility that naturally occurring arsenic could have been concentrated in the disposed water before it was released. As a result, arsenic is retained as a COPC at this site.

The results of the site and contaminant screening process are summarized in Table 6-1. Table 6-1 also lists all of the retained sites and all of the COPCs that will be analyzed at those retained sites.

6.3 Exposure Assessment

The human health exposure assessment quantifies the receptor intake of COPCs for select pathways. The assessment consists of estimating the magnitude, frequency, duration, and exposure route of COPCs to humans. To quantify the receptor intake, the following activities are performed as part of the BRA:

- Identification and characterization of exposed populations

- Evaluation of exposure pathways

- Estimation of contaminant concentrations at points of exposure for the following exposure pathways:

    - Soil pathway

    - Air pathway

    - Groundwater pathway

- Estimation of contaminant intakes.

Each of these activities is discussed in the following sections.

6.3.1 Identification and Characterization of Exposed Populations

The following current human populations could potentially be exposed to contaminants found at, or originating from, WAG 1:

6.3.1.1 Workers. Because WAG 1 is currently operational, workers at the site are potential receptors. The following two occupational exposure scenarios are analyzed in the BRA:

1. A current occupational scenario that lasts for 25 years from the present.

2. A future occupational scenario that starts in 100 years and lasts for 25 years.

6.3.1.2 Residents. For the purposes of the BRA, residential development will be considered as a potential future use of the site, and a future residential exposure scenario is quantitatively evaluated in the BRA.

For the purposes of the BRA, the assumption will be made that future residents will construct 3-m (10-ft) basements beneath their homes. As a result, all contamination detected in the upper 3 m (10 ft) of each release site will be evaluated for surface pathway exposures. This analysis method will hereafter be referred to as a "residential intrusion scenario," and all residential exposure scenario analysis in the OU 1-10 BRA will include the residential intrusion assumption.

The residential exposure scenario evaluated in the BRA considers a future resident who moves to the site in 100 years and lives there for 30 years. Because the nearest single-family residence is currently located several miles from the boundary of WAG 1, current residents are not to be evaluated in the BRA.

In general, the residential exposure scenario only evaluates adult exposures. The reason for this limitation is that the risk results presented in the BRA are calculated using very conservative exposure assumptions. These assumptions most likely cause the calculated risk results to overestimate the actual risks to even sensitive subpopulations, such as children, that would result from exposure to the site's contamination.

The exception to this rule is associated with the soil ingestion exposure route described in Section 6.3.3.1. Under this exposure route, six years of childhood soil ingestion and 24 years of adult soil ingestion are included in the contamination intake calculation. Soil ingestion is the most critical exposure route for children who may someday live at WAG 1 because of the relatively large amount of soil that children can ingest.

Groundwater pathway risks are calculated at 100 years in the future for use in the 100-year residential exposure scenario, but groundwater risks for each COPC are also calculated at the time of the maximum groundwater concentration of each COPC, as long as the maximum concentration occurs before 10,000 years in the future. See Section 6.3.3.3 for further discussion on the groundwater pathway analysis.

6.3.1.3 Future Land Use. TAN is currently composed of three active operations areas: the Contained Test Facility (CTF), the TSF and the WRRTF. A fourth area, the Initial Engine Test (IET) Area is

inactive and is scheduled for decontamination and decommissioning (D&D) over the next three years. Currently, 360 people are employed on projects located at TAN.

The CTF includes the Containment and Service Building [Loss-of-Fluid (LOFT)], and aircraft hanger, the Reactor Control and Equipment Building, and numerous support facilities. The major project now located at the CTF is the Specific Manufacturing Capability (SMC) which develops and produces armor for the U.S. Army and is located in the aircraft hanger. All portions of the CTF not involved in the SMC program are now maintained in shutdown condition or were inactivated in 1996.

TSF is the main administration, assembly, and maintenance area for TAN. Major programs now located at TSF include the Three Mile Island (TMI) Unit 2 Core Offsite Examination Program, the Process Experimental Pilot Plant (currently in shutdown condition), the Spent Fuel Program and portions of the SMC program.

The WRRTF includes the Thermal-Hydraulic Experimental Control Building, the Semiscale Control and Administration Building, and the Semiscale Assembly and Test Building. These facilities were used in support of the LOFT program that was conducted in the CTF. WRRTF now house a variety of small projects including one in which personnel are working on ways to test systems used to detect explosives that could continue for three to six years.

Unless it becomes necessary to resume former levels of defense related activities, the immediate future of TAN will consist of completing current programs, deactivating all facilities, and completing environmental restoration activities. Some of the facilities and utilities now supporting area programs and projects will be redirected to support these deactivation activities.

The 100-year future land use scenario projects a general continuation of the development of a more consolidated, secured facility that will continue the adaptive reuse of facilities adequately located for new Department of Energy (DOE) and/or non-DOE industrial uses (DOE-ID 1996). Over this 100-years it is anticipated that TAN will undergo a D&D program and be redeveloped as a non-nuclear industrial area.

6.3.2 Evaluation of Exposure Pathways

Once potentially exposed populations have been identified and characterized, exposure pathways can be traced from the site to the exposed populations. Each exposure pathway describes a mechanism by which a population or individual could be exposed to contaminants originating from one or more release sites at WAG 1. Only those exposure pathways deemed to be complete (i.e., where a plausible route of exposure can be demonstrated from the site to the receptor) are quantitatively evaluated in the BRA.

Based on information presented in the WAG 1 CSM (Figure 6-1), the following exposure scenarios, exposure pathways, and exposure routes will be evaluated in the BRA:

- Exposure scenarios

  - Occupational

  - Residential intrusion

- Exposure pathways

- Groundwater

- Air

- Soil

• Exposure routes

- Soil ingestion

- Inhalation of fugitive dust

- Inhalation of volatiles

- External radiation exposure

- Dermal absorption from soil (exposure route is screened from further consideration in Section 6.3.3.1)

- Groundwater ingestion (residential scenario only)

- Ingestion of home grown produce (residential scenario only)

- Dermal absorption of contaminants in groundwater (residential scenario only)

- Inhalation of volatiles from indoor use of groundwater (residential scenario only).

6.3.3 Estimation of COPC Concentrations at Points of Exposure

Exposure concentrations associated with each COPC are estimated for groundwater, air, and soil. The following sections provide details on how the estimated concentrations for each of these media are calculated.

Two tables that provide general information associated with estimation of COPC concentrations at points of exposure are included in Appendix B. Table B-30 shows the surface areas and soil volumes used in the BRA contaminant concentration calculations, and Table B-31 shows values assigned to various soil pathway and groundwater pathway parameters [molecular weights, radionuclide half-lives, soil to water partition coefficients (k_d), solubilities, octanol-water partition coefficients (K_{ow}), organic carbon partition coefficients (K_{oc}), diffusivities, Henry's Law Constants, and plant uptake factors (PUFs).

The depths of contamination evaluated for the exposure routes discussed in the following sections are based on guidance given in the *INEL Track-2 Investigation Manual* (DOE-ID 1994). Specifically, contaminant concentrations are based on the 95% upper confidence limit (UCL) on the mean concentrations (or maximum concentration if the maximum is less than the 95% UCL) of samples collected over the following depth ranges:

Depth	Exposure Route(s)
0 to 0.2 m (0 to 6 in.)	Occupational scenario: soil ingestion, inhalation of fugitive dust, inhalation of volatiles
0 to 1.2 m (0 to 4 ft)	Occupational scenario: external radiation exposure
0 to 3 m (0 to 10 ft)	Residential scenario: all soil pathway and air pathway exposure routes
All sample results included, regardless of depth	Residential scenario: all groundwater pathway exposure routes

For all depth ranges, 95% UCL concentrations of the mean are calculated as described in *EPA Guidance on Calculating Concentration Terms* (EPA 1992b). As part of the analysis, all data sets are assumed to have lognormal distributions. Tables B-32 through B-34 show the soil concentrations used in the BRA calculations by depth interval for each COPC.

The concentration values shown in Tables B-32 through B-34 indicate that a given COPC was detected in the depth interval shown in the table, not that the COPC contamination extends to the bottom of the interval. For example, chromium could have a calculated 0-to-3-m (0-to-10-ft) concentration at a given site shown in Table B-34 even if the site's chromium contamination only extends from 0 to 1.5 m (0 to 5 ft). The average concentrations for each of the above intervals were calculated by averaging detected concentrations in 0.3-m (1-ft) intervals. For example, the 0-to-1.2-m (0-to-4-ft) average concentrations were calculated by determining the 95% UCL on the mean or maximum concentration, whichever is less, for each contaminant in the 0-to-0.3-m (0-to-1-ft), 0.3-to-0.6-m (1-to-2-ft), 0.6-to-0.9-m (2-to-3-ft), and 0.9-to-1.2 m (3-to-4-ft).

In the exposure point concentration calculations, the only form of contaminant decay considered is radioactive decay (i.e., nonradionuclides are assumed to persist indefinitely in the environment). Radioactive decay is accounted for by estimating radionuclide concentrations at the start of a given exposure scenario, and then calculating the average concentrations that will exist during the length of the scenario. For example, the concentration of a given radionuclide analyzed in the current occupational exposure scenario is the average concentration that would exist between 0 and 25 years in the future, and the concentration analyzed in the 100-year future residential scenario is the concentration that would exist from 100 to 130 years. The average radionuclide concentrations over each time period are shown in Tables B-35 through B-39. The concentrations shown in these tables are used to calculate intakes for radionuclides as discussed in Section 6.3.4.

The effects of radioactive progeny are only considered by using "+D" SFs in the radionuclide risk calculations (see Section 6.5). Decay and in growth calculations are not performed for complete radionuclide decay chains. The use of "+D" SFs account for risks produced by daughter products that are in secular equilibrium with their parent radionuclides (EPA 1995).

6.3.3.1 Soil Pathway Methodology. Soil pathway risks are evaluated for each retained site. The following exposure routes are evaluated in the soil pathway analysis:

- Soil ingestion

- Ingestion of homegrown produce (residential scenario only)

- External radiation exposure

- Dermal absorption from soil [exposure route is insignificant for all COPCs and is not quantitatively evaluated in the BRA (see below)].

6.3.3.1.1 Soil Ingest Methodology—Because exposures through the soil pathway are not likely to occur from more than one release site at a time, the soil pathway is evaluated on a site-by-site basis. The possible exception to this rule is associated with the external radiation exposure route. Retained sites that have radionuclide contamination are evaluated to determine whether radiation produced by one site could affect a receptor located at an adjacent site (Section 6.6).

As with the groundwater and air pathways, soil pathway risks and HQs are calculated at 0 and 100 years in the future for the occupational exposure scenario, and at 100 years for the residential scenario.

6.3.3.1.2 Homegrown Produce Ingestion Methodology—The homegrown produce ingestion exposure route includes an evaluation of COPC concentrations in plants caused by both root uptake and irrigation with contaminated groundwater. At each retained site, the total source concentration evaluated in the homegrown produce ingestion exposure route is calculated by combining the 95% UCL on the mean concentration for a given COPC (or the maximum concentration if the maximum is less than the 95% UCL) with the soil concentration that would result from equilibrium partitioning between soil and groundwater contaminated with the COPC.

Homegrown produce concentrations assumed for each COPC are presented in Table B-40. To evaluate the average soil concentration of radioactive COPCs in soil when irrigating with groundwater, the integrated form of Equation 5.39 in *Nuclear Regulatory Commission (NRC) Guidance Document* (NRC 1993) is used:

$$C_s(t) = \frac{\frac{\dot{I}_v}{L_i + \lambda}\left(t_e + \frac{e^{-(L_i + \lambda)t_e}}{L_i + \lambda}\right) + \frac{C_{so}}{L_i + \lambda}\left(1 - e^{-(L_i + \lambda)t_e}\right) - \frac{\dot{I}_v}{(L_i + \lambda)^2}}{t_e} \quad (6\text{-}2)$$

where

$C_s(t)$	=	the average concentration of a COPC in soil for the exposure period, t_e (pCi/g)
$\dot{I}_v$	=	COPC input rate from irrigation (pCi/g-day)
L_i	=	leach rate constant (day)-1
λ	=	radioactive decay rate constant (day)-1
t_e	=	exposure period [10,950 day (30 years ' 365 days/year)]
C_{so}	=	average concentration of COPC in the top 3 m (10 ft) of soil at the start of the residential exposure period (pCi/g).

For nonradioactive COPCs, this equation reduces to the following:

$$C_s(t) = \frac{\dfrac{\dot{I}_v}{L_i}\left(t_e + \dfrac{e^{-(L_i t_e)}}{L_i}\right) + \dfrac{C_{so}}{L_i}\left(1 - e^{-(L_i t_e)}\right) - \dfrac{\dot{I}_v}{L_i^2}}{t_e} \qquad (6\text{-}3)$$

The COPC input rate from irrigation is given by the following equation:

$$\dot{I}_v = C_W \times \frac{IR}{\rho \times T} \qquad (6\text{-}4)$$

where

$\dot{I}_v$ =   COPC input rate from irrigation (mg/g-day or pCi/g-day)

C_W =   average concentration of a COPC in groundwater for the exposure period (mg/L or pCi/L)

I_R =   irrigation rate (8.47 L/m^2-yr × 90 days/365 yrs) (Maheras et al. 1994)

ρ =   soil density (1.5E+06 g/m^3)

T =   thickness of root zone (0.2 m) (7 in.) [International Atomic Energy Agency (IAEA) 1994].

The leach rate constant is given by the following equation (Baes and Sharp 1983):

$$L_i = \frac{P}{\theta_c \times \left(1 + \dfrac{K_d \times \rho}{\theta_c}\right) \times T} \times CF \qquad (6\text{-}5)$$

where

P =   net water percolation rate (0.86 m/1 year) [infiltration rate of 0.1 m/1 year, as presented in *INEL Track 2 Guidance* (DOE-ID 1994), plus the contribution from irrigation]

θ_c =   volumetric water content in source volume (0.41 m^3/m^3) (Rood 1994)

K_d =   COPC-specific soil-to-water partition coefficient (cm^3/g)

ρ =   soil density (1.5 g/cm^3)

T =   thickness of root zone (0.2 m) (IAEA 1994)

CF   =   conversion factor (1 year/365 days).

The radioactive decay constant is given by the following equation:

$$\lambda = \frac{\ln 2}{T_{1/2}} \tag{6-6}$$

where

$T_{1/2}$ =   half-life of a radionuclide (d).

Finally, concentrations of COPCs in affected homegrown produce are calculated using the following equation (EPA 1995):

$$C_p(t) = C_s(t) \times B_v \tag{6-7}$$

where

Cp(t)   =   average concentration of a COPC in homegrown produce from root uptake (pCi/g or mg/kg)

Cs(t)   =   average concentration of a COPC in soil for the exposure period (pCi/g or mg/kg)

Bv   =   COPC-specific soil-to-plant uptake coefficient (mass of COPC/dry mass of plant material per mass of COPC/dry mass of soil).

Homegrown produce contaminant concentrations calculated using the above equations are presented in Table B-40.

6.3.3.1.3 External Radiation Exposure Methodology—

For the external radiation exposure route, standard EPA protocols are used to estimate risks for all retained sites. In other words, external radiation exposure risks are calculated by multiplying radiation intakes for specific isotopes by the radionuclide SFs presented in EPAs Health Effects Assessment Summary Tables (HEAST) (EPA 1994). The standard EPA protocols are used because all of the retained sites in the BRA have radionuclide contamination that is at least 0.2 m (6 in.) thick over a large area. This thickness is large enough to satisfy the assumption that an increase in source thickness will not cause an increase in surface radiation exposures.

6.3.3.1.4 Dermal Exposure Methodology—

Risks from dermal absorption from soil are controlled by a contaminant's potential for being absorbed through skin. This potential is quantified by a contaminant's dermal absorption factor [i.e., the fraction of a given contaminant that can be absorbed through skin (ABS)]. ABS values are not well quantified for many of the contaminants that have been detected at WAG 1, however, EPA Region III has issued general guidelines for default ABS values (EPA 1995).

Organic contaminants have the greatest potential for producing unacceptable dermal absorption from soil exposures. The reason for this distinction is that, in general, organic contaminants have relatively high ABS values. For example, EPA 1995 recommends assuming an ABS value of 3% for volatile organic

contaminants with vapor pressures lower than benzene (i.e., vapor pressure less than 95.2 mm Hg), and an ABS value of 10% for semi-volatile contaminants. Since ABS values are poorly defined for most contaminants, and organic contaminants have the greatest potential for producing unacceptable dermal absorption risks, organic contaminants are quantitatively evaluated under the dermal absorption from soil exposure route in this BRA. The recommended ABS values from EPA (1995) are used for this evaluation.

The exception to the above rule involves the evaluation of arsenic. EPA (1995) recommends an arsenic ABS value of 3.2%. Since the arsenic ABS value is relatively high, and since arsenic may have been released at the TSF-07 Disposal Pond, arsenic is included in the dermal exposure analysis.

EPA (1995) also recommends that the following equation be used for calculating absorbed dose from dermal contaminants.

$$AD = \frac{C_{soil} \times SA \times AF \times ABS \times EF \times ED \times CF}{BW \times AT} \tag{6-8}$$

where

| AD | = | absorbed dose (mg/kg-d) |

| C_{soil} | = | contaminant soil concentration (mg/kg) |

| SA | = | skin surface area available for contact (3,000 cm^2/event) |

| AF | = | soil to skin adherence factor (0.5 mg/cm2) |

| ABS | = | absorption factor (unitless) |

| EF | = | exposure frequency (350 events/yr for residential exposures) |

| ED | = | exposure duration (30 years for residential exposures) |

| CF | = | conversion factor (1E–06 kg/mg) |

| BW | = | body weight (70 kg) |

| AT | = | averaging time [10,950 days for noncarcinogens (= 30 yr × 365 d/yr), or 25,550 days for carcinogens (= 70 yr × 365 d/yr)]. |

Absorbed dose for the dermal absorption exposure route is similar to contaminant intakes for other exposure routes (see Section 6.5). As a result, risks and HQs for dermal absorption exposures are calculated using the following equations:

$$Risk = \frac{AD \times SF}{GI} \tag{6-9}$$

where

Risk	=	contaminants specific carcinogenic risk (unitless)
AD	=	absorbed dose (mg/kg-d)
SF	=	contaminant specific oral slope factor $[(mg/kg-d)^{-1}]$.
GI	=	gastrointestinal absorption efficiency factor [= 0.05 (unitless) (EPA 1989)].

$$HQ = \frac{AD}{RfD * GI}$$ (6-10)

where

HQ	=	contaminant specific noncarcinogenic hazard quotient (unitless)
AD	=	absorbed dose (mg/kg-d)
RfD	=	contaminant specific oral reference dose (mg/kg-d).
GI	=	gastrointestinal absorption efficiency factor [= 0.05 (unitless) (EPA 1989)].

Table 6-2 lists all of the dermal contaminants that have been identified in WAG 1 surface soil sites. The table shows the site(s) where each contaminant has been detected, whether each contaminant is a volatile organic contaminants or a semi-volatile organic contaminants, each contaminant's vapor pressure, the assumed ABS value for each contaminant, and whether not each contaminant has available oral toxicity information that can be used to calculate dermal absorption risks. As shown in the table, benzo(a)pyrene and arsenic are the only WAG 1 dermal contaminants detected in surface soils that have available oral toxicity data. As a result, they are the only contaminants with calculated dermal absorption from soil risks and HQs. Table 6-3 shows the soil concentrations for benzo(a)pyrene and arsenic contaminant toxicity data, and the calculated dermal absorption from soil risks and HQ at the release sites.

Calculated dermal absorption from soil risks for benzo(a)pyrene and arsenic are within the NCP target risk range of 1E-06 to 1E-04 and the calculated dermal absorption HQ from arsenic at TSF-07 is less than the allowable HQ of 1. As a result, dermal absorption from soil risk will not be evaluated further in this BRA. Other organic contaminants may cause dermal absorption from soil exposures, but these contaminants do not have available toxicity information that can be used to calculate risk results. Potential impacts from dermal exposure to contaminants that do not have well defined ABS values, and exposures to contaminants that do not have available toxicity information are discussed further in the BRA uncertainty section (Section 6.6).

6.3.3.2 Soil Pathway Assumptions—The BRA soil pathway analysis includes the following assumptions:

- With the exception of the external radiation exposure route, soil pathway exposures from multiple release sites are insignificant.

- The likelihood that a future resident will raise meat and dairy products on a residential lot at WAG 1 is assumed to be negligible, in accordance with INEEL guidance on analysis of the homegrown produce ingestion exposure route (LMITCO 1996). As a result, risks from the ingestion of meat and dairy products are not quantitatively evaluated in the BRA.

- A receptor is assumed to be present at each retained site for the full exposure duration (30 years for a residential receptor and 25 years for an occupational receptor).

Table 6-2. Summary of dermal absorption from soil data for WAG 1 organic contaminants.

COPC	Inorganic, VOC, or SVOC	Site(s) Where Contaminant Has Been Detected	Vapor Pressure (mm Hg)	Assumed ABS (%)	Available Toxicity Data? (yes or no)
Arsenic	Inorganic	TSF-07	NA	3.2	Yes
benzo(a)pyrene	SVOC	TSF-07 TSF-36	5E–09	10	Yes
2-hexanone	VOC	WRRTF-01	2E+00	3	No
2-methylnaphthalene	VOC	TSF-03 WRRTF-01 WRRTF-13	1E+01	3	No
phenanthrene	SVOC	TSF-03 TSF-07 WRRTF-01 WRRTF-13	1E+00	10	No
phthalate	Unknown	WRRTF-13	Unknown	10	No
propionitrile	VOC	TSF-07	4E+01	3	No
n-propylbenzene	VOC	TSF-07	3E+00	3	No
tetrahydrofuran	Unknown	TSF-07	Unknown	10	No
tributylphosphate	Unknown	TSF-26	Unknown	10	No

Table 6-3. Calculated dermal absorption from soil risks and HQs.

Sites	Contaminant	0 to 10 ft Average Soil Concentration (see Table B-34) (mg/kg)	Oral Slope Factor (see Table B-64) (mg/kg-d)$^{-1}$	Oral Reference Dose (see Table B-64) (mg/kg-d)	Dermal Absorption From Soil Risk (unitless)	Dermal Absorption From Soil HQ (unitless)
TSF-07	Arsenic	1.43E+01	1.5E+00	3E-04	1E-04	6E-01
	Benzo(a)pyrene	1.05E-01	7.3E+00	NA	1E-05	NA
TSF-36	Benzo(a)pyrene	3.30E-02	7.3E+00	NA	1E-05	NA

6.3.3.3 Air Pathway Methodology. All retained sites that have contamination in the top 3 m (10 ft) of soil are assumed to have a contaminant source that can be released into the air pathway. The exposure routes that are evaluated as part of the air pathway analysis are as follows:

- Inhalation of fugitive dust

- Inhalation of volatiles.

Because there is a possibility that contamination from multiple sites can mix together within the air volume above WAG 1, the air pathway is analyzed in a cumulative manner in the WAG 1 BRA. To perform this cumulative analysis, a WAG-wide average soil concentration is calculated for each COPC. The concentration of each COPC in the respirable particulate matter above WAG 1 is assumed to equal this average soil concentration. Averaging contaminant concentrations above WAG 1 for the air pathway produces one contaminant-specific risk estimate for each air pathway exposure route [i.e., for each time period, each air pathway exposure route has the same risk or HI at every retained site (see Section 6.5)].

The equations discussed below will be used to estimate airborne contaminant concentrations:

$$C_{air} = CF \times R \times C_{soil} \tag{6-11}$$

where

C_{air} = contaminant concentration in air (mg/m3 or pCi/m3)

CF = conversion from kg to mg for nonradionuclides or g to mg for radionuclides

R = airborne respirable particulate matter concentration (0.018 mg/m3). Value is given in Appendix B of the *INEL Site Environmental Monitoring Reports* (e.g., Hoff et al. 1993), and represents the arithmetic mean, of weekly airborne respirable particulate matter concentrations by the TAN low volume air sampling station

C_{soil} = WAG average contaminant soil concentration (mg/kg or pCi/g) weighted by site area.

and

$$C_{soil} = \frac{\Sigma \ C_n \ A_n}{A_T} \tag{6-12}$$

where

C_n = contaminant soil concentration at site n (mg/kg or pCi/g)

A_n = surface area of site n (m^2)

6-27

A_T =   total area of the WAG 1 retained sites (m^2)

n   =   number of retained sites.

The equation used for estimating concentrations of airborne volatiles is as follows:

$$C_{air} = \frac{\Sigma (C_n / VF_n) A_n}{A_T} \tag{6-13}$$

where

C_n =   contaminant soil concentration at site n (mg/kg)

VF_n =   volatilization factor [as described in *INEL Track 2 Guidance* (DOE-ID 1994)] for site *n* (m^3/kg)

A_n =   surface area of site *n* (m^2)

A_T =   total area of the WAG 1 retained sites (m^2).

These equations produce conservatively high estimates of airborne COPC concentrations because no credit is taken for dilution of airborne concentrations caused by dust blown from uncontaminated areas of the WAG.

As with the soil pathway analysis, the air pathway receptor is either a current occupational worker (who is assumed to be exposed for 25 years) or a hypothetical future resident (who is exposed for 30 years). Air pathway risks and HQs are calculated at 0 and 100 years in the future for the occupational scenario, and at 100 years in the future for the residential scenario. Estimated concentrations of COPCs in fugitive dust and estimated concentrations of volatiles for each exposure period are presented in Table B-42.

6.3.3.3.1 Air Pathway Assumptions—The BRA air pathway analysis includes the following assumptions:

- The concentration of each retained contaminant in the respirable particulate matter above the WAG will be equal to each contaminant's WAG wide average soil concentration.

- The airborne concentration of each retained contaminant will be the same at every point inside the WAG boundaries.

- The air pathway receptor will be assumed to spend the entire exposure duration (25 years for current occupational workers and 30 years for future residents) working or living within the boundaries of the WAG.

6.3.3.4 Groundwater Pathway Methodology. To quantify risks for the future residential receptor (there is no occupational receptor for this exposure pathway), modeling of contaminant concentrations in groundwater is required. For the groundwater pathway analysis, every contaminant that is not eliminated

by the contaminant screening process (described in Section 6.2) is assumed to have the potential for migrating to groundwater, but only manmade sources of contamination are considered in the analysis. The following exposure routes are evaluated as part of the groundwater pathway analysis:

- Ingestion of groundwater

- Dermal absorption of groundwater

- Inhalation of volatiles produced by indoor use of groundwater.

WAG 1 includes two potential sources of groundwater contamination: contamination injected into the aquifer by the TSF-05 injection well, and contamination that could leach from surface and near surface soils. These two contaminant sources are evaluated as described in the following paragraphs. The only source of perched water at WAG 1 is the TSF-07 pond (see Section 4.1.10). This perched water is not expected to be a source of contamination that can impact groundwater beneath WAG 1 because the volume of water in the perched zone is small.

Contamination that has been injected into the aquifer by the TSF-05 injection well is being remediated under the OU 1-07B groundwater remediation project. According to the OU 1-07B ROD, the groundwater contaminants of concern in the TSF-05 contaminant plume are trichloroethylene, tetrachloroethylene, 1,2-dichloroethylene, Cs-137, H-3, Sr-90, and U-234. The remedial alternative selected in the ROD will reduce the plume's trichloroethylene concentration to 25 μg/L, tetrachloroethylene to 5 μg/L, 1,2-dichloroethylene to 70 μg/L, Cs-137 to 119 pCi/L [proposed maximum contaminant level (MCL)], H-3 to 20,000 pCi/L (MCL), Sr-90 to 8 pCi/L (MCL), and U-234 to 30 pCi/L (proposed MCL) by the beginning of the 100-year exposure scenario. This BRA assumes that the OU 1-07B remediation will be successful, and evaluates risks only from the residual contaminant concentrations listed above.

Groundwater concentrations resulting from surface and near surface sources are estimated using the computer code GWSCREEN (Rood 1994). For each COPC, GWSCREEN produces groundwater concentrations versus time as the codes output. From this output, the maximum 30-year average groundwater concentration of each COPC, and the 30-year average concentrations at 100 years in the future, are calculated. The average concentrations at year 100 are used to calculate groundwater pathway risks for the residential exposure scenario, and the maximum average concentrations are used to calculate maximum expected groundwater risks.

A period of 10,000 years is considered in the maximum groundwater risk analysis. As a result, if a COPC is expected to reach a maximum concentration after 10,000 years in the future, maximum groundwater pathway risks for the COPC are not evaluated. The 10,000-year limit is used to simplify the groundwater pathway analysis.

The total mass of each contaminant considered in the GWSCREEN modeling is calculated by summing the contaminant masses from the retained sites. The contaminant mass at each retained site is derived by multiplying the contaminant's 95% UCL of the mean concentration (or maximum concentration if the maximum is less than the 95% UCL) by the mass of contaminated soil at the site. For example, if a contaminant has a 95% UCL of the mean concentration of 5 mg/kg at three release sites with dimensions of 10 by 10 by 1 m (30 by 30 by 3 ft), the mass of the contaminant that would be used in the GWSCREEN modeling would be 2.3E+06 mg [(3 sites) $\times$ (5 mg/kg/site) $\times$ (10 m) $\times$ (10 m) $\times$ (1 m) $\times$ (1E+06 cm^3/m^3) $\times$ (1.5 g/cm^3) $\times$ (1E–03 kg/g) = 2.3E+06 mg]. Values assigned to various GWSCREEN input parameters

are shown in Table B-43, and the COPC masses used in the GWSCREEN modeling are shown in Table B-44. Other information about how GWSCREEN calculates groundwater concentrations is included in the *RI/FS Work Plan* (Lewis et al. 1996).

Three input parameters shown in Table B-43 (length of source parallel to flow, width of source perpendicular to flow, and thickness of source) are based on the site dimensions shown in Table B-30. The length and width values are calculated by taking the square root of the total retained site surface area (shown at the bottom of Table B-30), and the thickness value is the average depth of contamination at the retained sites.

Appendix C contains the GWSCREEN output files for each COPC, and Table B-45 and B-46a summarize the results of the GWSCREEN runs. Because the retained site sources are combined for the GWSCREEN modeling, the GWSCREEN output concentrations are not projected to occur at any specific point beneath the WAG. As with the air pathway analysis, the GWSCREEN results are assumed to be conservative estimates of the maximum groundwater concentrations that might occur at any point beneath the WAG during the residential exposure scenario.

The contaminant concentrations shown in Tables B-45 and B-46a are expected to overestimate the true aquifer concentrations that will be produced by infiltration of contaminants at WAG 1. Because of the great complexity of the subsurface beneath the WAG and limited information about factors that influence flow and transport of contaminants in groundwater, the uncertainty about potential contaminant concentrations associated with the groundwater pathway exposure routes is greater than the uncertainty associated with any other exposure pathway in this BRA. To compensate for this relatively large uncertainty, conservative assumptions are used throughout the groundwater pathway analysis. Some of the conservative assumptions that are used in the GWSCREEN analysis are as follows:

- All infiltration into the WAG is assumed to occur through contaminated areas of the WAG. No credit is taken for infiltration into areas between retained sites.

- GWSCREEN uses a plug flow model for contaminant transport through the unsaturated zone. This model does not take any credit for contaminant dispersion in the unsaturated zone.

- Groundwater flow through fractured basalt in the unsaturated zone is assumed to occur very rapidly in comparison to flow through sedimentary material. This assumption is incorporated into the GWSCREEN modeling by using a depth to the aquifer that is only 1/10th of the total unsaturated zone thickness beneath WAG 1. Using this small depth results in a relatively short unsaturated zone travel time in which radioactive decay can occur. As a result, the GWSCREEN estimates of radionuclide concentrations are expected to be conservatively high. Since no loss mechanisms are assumed to be present for nonradioactive contaminants, the only affect that the small unsaturated zone thickness assumption has on these contaminants is that it reduces the time at which the contaminants are predicted to reach the aquifer. The assumption has no affect on the predicted contaminant concentrations in the aquifer after the contaminants have reached the saturated zone.

- All COPC mass contained in surface soils at the WAG is assumed to contribute to groundwater contamination. For the purposes of the GWSCREEN modeling, no credit is taken for loss of COPC mass caused by mechanisms such as wind erosion, surface water erosion, contaminant uptake into plants, etc. The only contaminant loss mechanism that is considered in the groundwater pathway evaluation is radioactive decay.

- Estimates of COPC mass that may be transported to groundwater are based on upper limit estimates of COPC soil concentrations.

Two other conservative assumptions that are included in the groundwater analysis, but not limited to the GWSCREEN modeling, are as follows:

- The groundwater receptor is assumed to take all drinking water from a well located at the center of the equivalent rectangle's downgradient edge for 30 years.

- All contaminants are assumed to be uniformly distributed within the groundwater modeling source volume.

The only source of perched water known to exist at WAG 1 lies beneath the TSF-07 disposal pond (see Section 4.1.10.2). The perched water body is present because of the continuing disposal of water in the TSF-07 pond. Once the water disposals are discontinued, the perched water body is expected to dissipate.

Risks from ingestion of water taken from the TSF-07 perched water body are not calculated in this BRA for three reasons. First, the water body is present as a result of water disposals to the TSF-07 pond. These disposals will be discontinued long before the end of the 100 year INEEL institutional control period, so it's unlikely that anyone will be able to drill a drinking water well into the perched water body. Second, the TSF-07 perched water body is relatively small, so it's unlikely that the body could produce enough water to support a residence over an extended period of time. Third, the TSF-07 disposal pond is permitted for land application of waste water with the State of Idaho. Part of the permitting process for the disposal pond was an evaluation of risks associated with contaminants that might be contained in the disposed water. The permit was issued because these risks are acceptable. Much of the contamination contained id the disposed water would be deposited in the pond's sediments, so the conclusion that risks associated with the perched water are acceptable.

6.3.3.5 Dermal Absorption From Groundwater Methodology. Exposures to COPCs through dermal absorption of groundwater are controlled by a given contaminants permeability coefficient of water through skin (K_p^w). According to EPA guidance (EPA 1992b), if the permeability coefficient for a given COPC is less than 0.1 cm/hour, then the dermal absorption from groundwater exposure route produces risks that are less than risks produced by the groundwater ingestion exposure route for that COPC. In the BRA, the default permeability coefficient used for inorganic COPCs is 1E–03 cm/hour, and the permeability coefficients for organic COPCs are estimated using the following equation:

$$\text{Log } K_p^w = -2.72 + 0.71 \text{ Log } K_{ow} - 0.0061 \text{ MW} \tag{6-14}$$

where

$\quad$ Kow $\quad = \quad$ octanol/water partition coefficient (unitless)

$\quad$ MW $\quad = \quad$ molecular weight (g/mol).

Permeability coefficients for WAG 1 COPCs are shown in Table B-41. Because many of the organics have permeability coefficients that are greater than the screening level of 0.1 cm/hour, the dermal

absorption from the groundwater exposure route is quantitatively evaluated in the BRA. Contaminant intakes for this exposure route are calculated using the equations shown in Section 6.3.4.

In the BRA, exposures caused by the inhalation of water vapors from indoor water use are calculated based on experimental data derived from a study of household water contaminants (Andelman 1990). This study derived a volatilization constant that defines the relationship between the concentration of a contaminant in household water and the average concentration of the volatilized contaminant in air. In the derivation, all uses of household water were considered (e.g., showering, laundering, and dish washing), and certain reasonable assumptions were made in deriving a volatilization fraction. For example, the study included assumptions about water usage for a family of four, the volume of the dwelling, and the air exchange rate. Furthermore, the study assumed that the average transfer efficiency weighted by the type of water use is 50% (i.e., half of the concentration of each chemical in water will be transferred into air by all types of water uses).

In the BRA indoor water use analysis, a central tendency value [6.50E–02 mg/m^3 air per mg/L water (Andelman 1990)] for the volatilization fraction of a COPC is used to develop estimates of COPC airborne concentrations. The airborne concentrations are calculated by multiplying the central tendency value by the COPC groundwater concentrations shown in Table B-45. These concentrations are then used to develop contaminant intake estimates using the equations shown in Section 6.3.4. The estimates of COPC airborne concentrations from indoor water use calculated for the BRA are shown in Table B-46b.

6.3.4 Estimation of Contaminant Intakes

The general equation that is used to calculate intakes for most of the BRA exposure routes is as follows (EPA 1989):

$$Intake = \frac{C \times IR \times EF \times ED}{BW \times AT} \tag{6-15}$$

where

Intake $\quad=\quad$ contaminant intake (mg/kg-day)

C $\quad=\quad$ concentration of a given contaminant in a contaminated medium (soil, air, water, etc.) (mg/kg, mg/m^3, mg/L, etc.)

IR $\quad=\quad$ ingestion rate of the contaminated medium (mg/day, m^3/day, L/day, etc.)

EF $\quad=\quad$ exposure frequency (day/year)

ED $\quad=\quad$ exposure duration (year)

BW $\quad=\quad$ body weight (kg)

AT $\quad=\quad$ averaging time (day).

The above equation applies to all exposure routes except exposure to external radiation. For the external radiation exposure route, intakes are calculated using the following general equation:

$$Intake = C \times ET \times EF \times ED \times CF \qquad (6\text{-}16)$$

where

Intake	=	radiation intake (pCi-year/g)
C	=	radionuclide concentration in soil (pCi/g)
ET	=	exposure time (hour/day)
EF	=	exposure frequency (day/year)
ED	=	exposure duration (year)
CF	=	conversion factor (1.14E–04 year/hour).

The specific intake factor equations used for the BRA exposure routes are shown in Tables B-47 through B-57. Results of the BRA intake factor calculations are shown in Tables B-58 through B-63.

6.4 Toxicity Assessment

This section provides the toxicity constants that will be used for risk characterization purposes and summarizes toxicological information for the WAG 1 radioactive and nonradioactive COPCs. For this assessment, and consistent with *EPAs RAGS* (EPA 1989a), the toxicity information is summarized for two broad categories of potential effects: noncarcinogenic and carcinogenic effects. These two categories are selected because of the slightly differing methodologies for estimating potential health risks associated with exposures to carcinogens and noncarcinogens.

The toxicity constants used in the BRA are obtained from several sources. The primary source of information is the EPAs Integrated Risk Information System (IRIS). IRIS contains only those toxicity constants that have been verified by EPAs Reference Dose or Carcinogen Risk Assessment Verification Endeavor Work Groups. The IRIS database is updated monthly and supersedes all other sources of toxicity information. If the necessary data are not available in IRIS, EPAs HEAST (EPA 1994) are used. The toxicity constant tables are published annually and updated approximately twice per year. HEAST contains a comprehensive listing of provisional risk assessment information that has been reviewed and accepted by individual EPA program offices, but has not had enough review to be recognized as high-quality, agency-wide information (EPA 1994).

6.4.1 Toxicity Assessment for Carcinogenic Effects

Potential carcinogenic risks are expressed as an estimated probability that an individual might develop cancer from lifetime exposure. This probability is based on projected intakes and chemical-specific dose-response data called cancer SFs. Cancer SFs and the estimated daily intake of a compound, averaged over a lifetime of exposure, are used to estimate the incremental risk that an individual exposed to that compound may develop cancer. This estimate is derived using the following equation:

$$Risk = Intake \times SF \qquad (6\text{-}17)$$

where

Risk	=	Carcinogenic risk (unitless)
Intake	=	Contaminant intake (mg/kg-day or pCi)
SF	=	Slope factor ([mg/kg-day]-1 or [pCi]-1).

There are two classes of potential carcinogens identified at WAG 2 release sites: chemical carcinogens and radionuclides. These two classes of carcinogens are discussed separately in the following subsections.

6.4.1.1 Toxicity Assessment for Chemical Carcinogens

6.4.1.1 *Toxicity Assessment for Chemical Carcinogens*. Evidence of chemical carcinogenicity originates primarily from two sources: (1) lifetime studies with laboratory animals and (2) human (epidemiological) studies. For most chemical carcinogens, animal data from laboratory experiments represent the primary basis for the extrapolation. Major assumptions arise from the necessity of extrapolating experimental results: across species (i.e., from laboratory animals to humans); from high-dose regions (i.e., levels to which laboratory animals are exposed) to low-dose regions (i.e., levels to which humans are likely to be exposed in the environment); and across routes of administration (i.e., inhalation versus ingestion). Federal regulatory agencies have traditionally estimated human cancer risks associated with exposure to chemical carcinogens on the administered-dose basis according to the following approach:

- The relationship between the administered dose and the incidence of cancer in animals is based on experimental animal bioassay results.

- The relationship between the administered dose and the incidence of cancer in the low-dose range is based on mathematical models.

- The dose-response relationship is assumed to be the same for both humans and animals, if the administered dose is measured in the proper units.

Effects from exposure to high (i.e., administered) doses are based on experimental animal bioassay results, while effects associated with exposure to low doses of a chemical are generally estimated from mathematical models.

For chemical carcinogens, EPA assumes a small number of molecular events can evoke changes in a single cell that can lead to uncontrolled cellular proliferation and tumor induction. This mechanism for carcinogenesis is referred to as stochastic, which means that there is theoretically no level of exposure to a given chemical that does not pose a small, but finite, probability of generating a carcinogenic response.

Because risk at low exposure levels cannot be measured directly either in laboratory animals or human epidemiology studies, various mathematical models have been proposed to extrapolate from high to low doses (i.e., to estimate the dose-response relationship at low doses). The three most frequently used models are (1) the one-hit model, (2) the log-probit model, and (3) the multistage model. The one-hit model is based on the premise that a single molecule of a contaminant can be the single event that precipitates tumor induction (Cornfield 1977). In other words, there is some finite response associated with any exposure. The log-probit model assumes that a response is normally distributed with the logarithm of the dose (Mantel et al. 1971). This theory seems to have little scientific basis, although some physiological

parameters are lognormally distributed. This model usually yields much lower potency estimates because of the implied threshold at lower doses.

Currently, regulatory decisions are based on the output of the linearized multistage model (EPA 1989a). The basis of the linearized multistage model is that multiple events (versus the single-event paradigm of the one-hit model) may be needed to yield tumor induction. The linearized multistage model reflects the biological variability in tumor frequencies observed in animals or human studies (Crump, Guess, and Deal 1977). The dose-response relationship predicted by this model at low doses is essentially linear. Use of this model provides dose-response estimates intermediate between the one-hit and the log-probit models. It should be noted that the SFs calculated for nonradiological carcinogens using the multistage model represent the 95th percentile UCL on the probability of a carcinogenic response. Consequently, risk estimates based on these SFs are conservative estimates representing upper-bound estimates of risk where there is only a 5% probability that the actual risk is greater than the estimated risk.

Most models produce quantitatively similar results in the range of observable data, but yield estimates that can vary by three or four orders of magnitude at lower doses. Animal bioassay data are simply not adequate to determine whether any of the competing models are better than the others. Moreover, there is no evidence to indicate that the precision of low-dose risk estimates increases through the use of more sophisticated models. Thus, if a carcinogenic response occurs at the exposure level studied, it is assumed that a similar response will occur at all lower doses, unless evidence to the contrary exists.

Uncertainties in the toxicity assessment for chemical carcinogens are dealt with by classifying each chemical into one of several groups, according to the weight of evidence from epidemiological studies and animal studies, as follows:

- Group A—Human carcinogen (sufficient evidence of carcinogenicity in humans)

- Group B—Probable human carcinogen (B1–limited evidence of carcinogenicity in humans; B2–sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans)

- Group C—Possible human carcinogen (limited evidence of carcinogenicity in the animals and inadequate or lack of human data)

- Group D—Not classifiable as to human carcinogenicity (inadequate or no evidence)

- Group E—Evidence of noncarcinogenicity for humans (no evidence of carcinogenicity in adequate studies).

Table B-64 provides the SFs, in $(mg/kg/day)^{-1}$, and the weight-of-evidence for each WAG 1 COPC.

SFs for polynuclear aromatic hydrocarbons (PAHs) are derived somewhat differently than SFs for other chemical carcinogens. Specifically, the methodology for estimating carcinogenic potencies of PAHs outlined in EPAs Environmental Criteria and Assessment Offices *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons* (EPA 1993) was followed. The guidance uses a toxicity weighting factor approach to estimate the oral cancer SFs for several PAHs relative to the SF for benzo(a)pyrene. This methodology was developed because, while EPA has classified seven PAHs as

Group B2-probable human carcinogens, data are sufficient to accurately measure dose-response (and by extension calculate SF) only for benzo(a)pyrene.

To obtain an estimate of total carcinogenic risk resulting from modeled exposures to carcinogens at the site, cancer risks are summed across all exposure routes for all carcinogens. Cancer risks from exposure to multiple carcinogens across multiple pathways are assumed to be additive, based on EPA carcinogen risk assessment guidelines (EPA 1986).

6.4.1.2 Toxicity Assessment for Radionuclides. An extensive body of literature exists that describes the health effects of radionuclides on humans and animals. Intensive research by national and international commissions has resulted in the establishment of widely accepted limits to which workers and the public may be exposed without clinically detectable effects. This literature has resulted in EPA classifying all radionuclides as Group A carcinogens because radionuclides emit ionizing radiation, which, at high doses, has been associated with increased cancer incidence in humans. Human epidemiological data collected from the survivors of the Hiroshima and Nagasaki bomb attacks form the basis for the most recent extrapolation put forth by the National Academy of Science (NAS 1980). Conversely, for most nonradiological carcinogens, animal data from laboratory studies represent the primary basis for the extrapolation.

Another fundamental difference between the assessment of potential toxicity associated with exposure to radionuclide and nonradionuclide carcinogens is that SFs for radionuclides are typically best estimates (mean or median values rather than upper 95th-percentile values). Furthermore, in the past, risk factors for radionuclides have generally been based on fatalities (i.e., the number of people who actually died from cancer), while SFs for nonradiological carcinogens are based on incidence (i.e., the number of people who developed cancer). Finally, the SFs for radionuclides are expressed in different units, i.e., risk per $(pCi)^{-1}$ rather than $(mg/kg/day)^{-1}$.

Table B-64 lists SFs for all radionuclides identified at WAG 1 release sites. These nonthreshold SFs account for the following: the amount of radionuclide transported into the bloodstream, the decay of radioactive progeny within the body, the distribution and retention of the radionuclide and its progeny (if any) in the body, the radiation dose delivered to specific organs and tissues, and the age and sex of the exposed individuals (EPA 1994).

6.4.2 Toxicity Assessment for Noncarcinogenic Effects

Potential noncarcinogenic effects are evaluated by comparing daily intakes with chronic RfDs developed by the EPA. This section provides a definition of an RfD and discusses how it is applied in the OU 1-10 BRA. Table B-64 provides the RfD values for each of the COPCs identified at WAG 1 release sites.

A chronic RfD is an estimate of the daily exposure that can be incurred during a lifetime, without an appreciable risk of a noncancer effect being incurred in human populations, including sensitive subgroups (EPA 1989a). The RfD is based on the assumption that thresholds exist for noncarcinogenic toxic effects (e.g., liver or kidney damage). It is a benchmark dose operationally derived by the application of one or more order-of-magnitude uncertainty factors to doses thought to represent a lowest or no-observed-adverse-effect level (NOAEL) in humans. Thus, there should be no adverse effects associated with chronic daily intakes below the RfD value. Conversely, if chronic daily intakes exceed this threshold level, there is a potential that some adverse noncarcinogenic health effects might be observed in exposed individuals.

RfDs or SFs have not been developed by the EPA for the dermal exposure route. In the absence of these factors, the common practice has been to use the available toxicity measures for the oral route of exposure. This approach has been adopted in the BRA.

In evaluating the dermal pathway, the EPA recommends expressing chemical intake as absorbed dose and adjusting the oral toxicity measures to reflect absorbed dose (EPA 1989a). In deriving such values, consistency is required between the type of dose that forms the basis of the oral toxicity and the type of dose that will be calculated by the dermal exposure models. Specifically, a distinction must be made between an administered dose or intake (i.e., the amount of chemical taken into the body) and the absorbed dose (i.e., the amount of chemical that crosses the body membranes and enters the bloodstream). Most of the toxicity measures available from the EPA are expressed as administered dose (i.e., intake) rather than dose at the tissue level (i.e., absorbed dose). The adjustment of the oral toxicity measure can be accomplished only if sufficient data are available in the principal laboratory studies or on the oral absorption efficiency in the species on which the toxicity measures are based. EPA notes that exposure estimates for absorption efficiency should not be adjusted if the toxicity values are based on administered doses (EPA 1989a).

For risk characterization purposes, the potential health effects of chronic exposure to noncarcinogenic compounds are assessed by calculating an HQ for each COPC. An HQ will be derived by dividing the estimated daily intake by a chemical-specific RfD as shown in the following equation:

$$HQ = Intake \, / \, RfD \qquad\qquad (6\text{-}18)$$

where

HQ	=	hazard quotient (unitless)
Intake	=	contaminant intake (mg/kg-day).
RfD	=	reference dose (mg/kg-day)

An HQ greater than 1.0 indicates that exposure to a given contaminant (at the concentrations and for the duration and frequencies of exposure estimated in the exposure assessment) may cause adverse health effects in exposed populations. However, the level of concern associated with exposure to noncarcinogenic compounds does not increase linearly as HQ values exceed 1.0. In other words, HQ values do not represent a probability or a percentage. For example, an HQ of 10 does not indicate that adverse health effects are 10 times more likely to occur than an HQ value of 1.0. All one can conclude is that HQ values greater than 1.0 indicate that noncarcinogenic health impacts are possible and that the higher the HQ value, the greater the concern about potential adverse health effects.

Consistent with RAGS, chemical-specific HQs are summed across exposure routes to calculate a HI for each COPC. Individual pathway HI values are then summed to determine a cumulative HI value for all exposure pathways and COPCs at each release site. This approach may result in a situation where a total HI value for a given release site may exceed 1.0 even though none of the chemical-specific HQ values at the release site exceed 1.0.

6.4.3 Toxicity Profiles

The following subsections present general and contaminant-specific information on health effects relating to the COPCs evaluated in the BRA. All information presented in these subsections is from IRIS unless otherwise specified. Chemical-specific toxicity values for each COPC discussed in these subsections are presented in Table B-64.

6.4.3.1 Polycyclic Aromatic Hydrocarbons. PAHs are a group of chemicals formed during the incomplete burning of organic substances. PAHs can occur because of either anthropogenic or natural activity (e.g., forest fire). All of the carcinogenic PAHs of concern for WAG 1 are classified as Group B2 carcinogens except for phenanthrene and pyrene (weight-of-evidence = D). Some PAHs produce tumors in mice when administered orally, dermally, or by subcutaneous injection and have been shown to be mutagenic. Lung cancers have been shown to be induced in humans by exposure to PAH mixtures, including coal tar, coke-oven emissions, and cigarette smoke, which include benzo(a)pyrene and benzo(a)anthracene. An oral SF has been determined only for benzo(a)pyrene, which has been shown to be carcinogenic and genotoxic in animals. The oral SF for benzo(a)pyrene is 7.3 $(mg/kg\text{-}day)^{-1}$ (EPA 1993). All other SFs for carcinogenic PAHs of concern are derived from benzo(a)pyrene and presented in Table B-64. IRIS provides profiles suitable for summary of noncarcinogenic PAHs of concern. These profiles are provided in the following subsections.

PAHs tend to sorb strongly to soil and organic matter. Higher molecular weight PAHs tend to have lower solubilities in water. Hydrophobic PAHs have a high affinity for binding to organic matter and have relatively high biotransformation rates. Although most of the pyrene that partitions into water is likely to sorb onto organic matter, biodegradation of pyrene in water can be an important degradative pathway. The dominant mechanism of PAH removal from soil is microbial degradation. PAHs can persist in soils for years. The low solubility, low vapor pressure, and high octanol-water partition coefficient of pyrene results in its partitioning mainly between soil, with a small fraction partitioning into water and air.

6.4.3.1.1 Benzo(a)anthracene—See the general discussion for PAH in Section 6.4.3.1.

6.4.3.1.2 Benzo(b)fluoranthrene—See the general discussion for PAH in Section 6.4.3.1.

6.4.3.1.3 Naphthalene—Oral and subcutaneous administration of naphthalene did not cause cancer in rats. Intraperitoneal injection of 395 mg/kg-day of naphthalene in rats did not cause adverse fetal or maternal effects (Hardin et al. 1981). Ocular toxicity is the most common effect associated with short-term, high-level exposures in animals. Rabbits fed 1,000 mg/kg-day for 46 days experienced cataracts and retinopathy (Ghetti and Mariana 1956). Bronchial necrosis was observed in mice following a single intraperitoneal injection of 128 mg/kg (Mahavei, Bank, and Harvey 1977). Dogs experienced a 98% decrease in hemoglobin levels after receiving an oral dose of 1,800 mg/kg-day for 5 days (Zvelzer and Apt 1949). Mice fed 5.3 to 133 mg/kg-day for 90 days did not experience adverse effects (Shopp et al. 1984).

6.4.3.1.4 Acenaphthene—In a study of the effects of acenaphthene on mice, doses of 0, 175, 350, or 700 mg/kg/day of acenaphthene were given by gavaging to four groups of CD-1 mice (20 per sex per group) for 90 days (EPA 1989b). The toxicological evaluations of this study conducted by Hazelton Laboratories, Inc., for the Office of Solid Waste in Washington, D.C., included body-weight changes, food consumption, mortality, clinical pathological evaluations (including hematology and clinical chemistry), organ weights and histopathological evaluations of target organs. The results of this study indicated no treatment-related effects on survival, clinical signs, body-weight changes, total food intake, and ophthalmological alterations. Liver-weight changes accompanied by microscopic alterations (cellular

hypertrophy) were noted in both mid- and high-dose animals and seemed to be dose-dependent. Additionally, high-dose males and mid- and high-dose females showed significant increases in cholesterol levels. Although increased liver weights, without accompanying microscopic alterations or increased cholesterol levels, were also observed at the low dose, this change was considered to be adaptive and was not considered adverse. The lowest-observed-adverse-effect-level (LOAEL) is 350 mg/kg/day based on hepatotoxicity); the NOAEL is 175 mg/kg/day.

6.4.3.2 Phthalate Esters. Toxicity information for phthalate esters is discussed in the following sections.

6.4.3.2.1 Di-n-octylphthalate—Although relatively little specific information concerning di-n-octylphthalate is available, the environmental transport and fate of this contaminant can be largely inferred from data for phthalate esters as a group. Di-n-octylphthalate probably hydrolyzes in surface waters, but at such a slow rate that this process would not be significant under most conditions. Photolysis and oxidation do not appear to be important environmental fate processes (EPA 1985).

6.4.3.3 Polychlorinated Biphenyls (Aroclor 1260). Aroclor 1260 belongs to the class of compounds known as PCBs. PCB mixtures are classified as Group B2 carcinogens. Data on carcinogenicity in humans following exposures to PCBs are inadequate because of confounding exposures or lack of exposure quantification (EPA 1993). Exposure to commercial PCB mixtures has been shown to cause hepatocellular cancer in rats and mice, while most genotoxic and mutagenic bioassays with PCBs have been negative. The oral SF for PCBs (as a mixture) is $7.70E+00$ (mg/kg/day)$^{-1}$ (EPA 1994).

6.4.3.4 Dichlorodifluoromethane. In a study by Haskell Laboratory of long-term effects of dichlorodifluoromethane, rats and dogs received 300 ppm or 3,000 ppm of dichlorodifluoromethane in the diet for 2 years (Sherman 1974). Additionally, carcinogenic and three-generation reproductive studies were conducted in rats. Clinical biochemical, urine analytical, hematological or histopathological evaluations were performed. Except for decreased weight gain in rats (about 20% in females) receiving 3,000 ppm (150 mg/kg/day) dichlorodifluoromethane, no adverse effects were attributed to this compound in either rats or dogs.

This study is sufficiently complete to derive an RfD for adequate protection against adverse human health effects. The high dose (3,000 ppm or 150 mg/kg/day) caused decreased body weights in rats and is, therefore, considered a LOAEL, whereas the low dose (300 ppm or 15 mg/kg/day) in rats produced no adverse effects attributable to the oral administration of dichlorodifluoromethane, and is the NOAEL.

The Haskell Laboratory study is a chronic oral study of two species that incorporated extensive clinical and toxicologic parameters. Therefore, a high level of confidence in the study is appropriate. Confidence in the database is medium because of the lack of teratogenic and reproductive data; therefore, confidence in the RfD is also medium.

6.4.3.5 Metals. Toxicity information for metal COPCs is discussed in the following sections.

6.4.3.5.1 Arsenic—Acute exposure to arsenic causes severe throat irritation, gastrointestinal disturbance, and muscle spasms. This is followed by vertigo, delirium, and coma. Facial edema may also be evident. Sensory loss and hematopoietic symptoms associated with acute exposure are usually reversible. Chronic exposure, either by ingestion or inhalation, is marked by malaise and fatigue. Changes in the skin include hyperkeratosis. Anemia and neuropathy, liver injury, and "blackfoot disease" also result from chronic exposure.

Arsenic is a known carcinogen in humans. Ingestion is associated with increased incidence of skin cancer; lung cancer results from inhalation. Insufficient data exist to determine carcinogenic effects in animals.

6.4.3.5.2 Barium—Barium is distributed all over the earth and occurs most frequently as barite. Barium is used as a carrier for radium; a deoxidizer for copper; a lubricant for anode rotors in x-ray tubes; in paints, soap, paper, and rubber; in the manufacture of ceramics and glass; and as a heat stabilizer for plastics. Barium metal in the free state does not occur in nature. It is found in zinc or iron ores. It is emitted mostly by industrial processes involved in the mining, refining, and production of barium and barium-based contaminants and as a result of the combustion of coal and oil. Barium is taken up, retained, and excreted in mammals in much the same way as calcium compounds.

6.4.3.5.3 Chromium—IRIS contains no exposure information on chromium. Chromium exists as an ore in nature and is most widely used in steel manufacturing, plating, paint and pigment manufacturing, and leather tanning. While chromium exists in several valence states, only the trivalent form is an essential element for humans, and plays an important role in glucose and lipid metabolism. A deficiency is characterized by symptoms similar to diabetes mellitus and produces aortic plaques in rats. A deficiency of trivalent chromium also increases the toxicity of lead.

Intake of chromium in all forms occurs monthly through the water supply. The body stores chromium in skin, lungs, muscle, and fat. Hexavalent chromium is toxic, but is converted to trivalent chromium in the skin when present in small concentrations. Exposure to high concentrations of the hexavalent form induces dermatitis, inflammation of the larynx and liver when inhaled, and renal tubular necrosis.

The EPA has listed the LOAEL for chromium (III) and (VI) as 0.002 mg/m^3 (as chronic acid) (EPA 1991). The LOAEL is 7.14E–04 mg/m^3. The inhalation reference concentration (RfC) applicable to both forms of chromium is 2.00E–05 mg/m^3, with an associated uncertainty factor of 30. The oral RfD is 5.00E–03 mg/kg/day for hexavalent chromium and 1.0 mg/kg/day for trivalent chromium with an associated uncertainty factor of 100.

Only hexavalent chromium is considered a carcinogen because it may induce bronchiogenic carcinoma. A SF for inhalation of 4.20E+01 (mg/kg/day)$^{-1}$ is recommended for evaluations of carcinogenic effects.

High rates of lung cancer have been associated with chromate-producing industry workers. Chromate salts are carcinogenic in rats exposed by inhalation.

6.4.3.5.4 Manganese—IRIS contains current risk-based exposure information on manganese. Manganese is a human nutrient, serving as an enzyme cofactor supporting basal metabolism in both plants and animals. Manganese is rated as one of the least toxic of the trace elements and is not a human carcinogen. In drinking water, manganese is more bioavailable than when it is consumed in food. Foods with high levels of manganese are cereals, nuts, and green leafy vegetables.

Manganese poisoning does occur as the result of prolonged inhalation of airborne metal-laden dust. This results in neurological and psychological symptoms including irritability, motor impairment, speech disturbances, and compulsive behavior. Prolonged effects are similar to Parkinson's Disease. Very high doses result in nerve degeneration.

6.4.3.5.5 Mercury—The chemistry of mercury in the environment is complex, not only because of its various oxidation states but also because of biotic and abiotic methylation and demethylation processes, complexation with organic and inorganic ligands, and the differential solubility and volatility of various forms. Speciation is a major determinant of the fate, bioavailability, absorption, and toxicologic characteristics of mercury compounds. Lack of knowledge of the state of the mercury in WAG 1 soils is a large source of uncertainty in the exposure assessment for the contaminant.

Although the generally more toxic organic forms of mercury are unlikely to persist in the environment, they (in particular, methylmercury) may be formed in biotic tissues and are known to biomagnify through ecosystems, particularly aquatic systems (Wren 1986; Scheuhammer 1987).

Because of its chemical stability and lipophilicity, methylmercury readily penetrates the blood-brain barrier. The central nervous system is thus a major target organ in both mammals and birds. However, reproductive effects have been reported at even lower doses. Methylmercury can be converted to inorganic mercury both in tissues and by microflora in the gut. The homolytic cleavage of the mercury-carbon bond leads to generation of reactive intermediates, e.g., methyl and metal radicals, which cause cellular damage (Wren 1986; Scheuhammer 1987; Manzo et al. 1992).

6.4.3.6 Radionuclides. The EPA classifies all radionuclides as Group A carcinogens because radionuclides emit ionizing radiation and because of the extensive weight-of-evidence provided by epidemiological studies of radiation-induced cancers in humans. Ionizing radiation has sufficient energy to interact with matter and produce an ejected electron and a positively charged ion. In addition, ionizing radiation can produce new chemical species, known as free radicals, from water in the body. Free radicals are highly reactive and may combine with other elements or compounds within a cell to produce toxins or otherwise disrupt a cell's chemical balance. These disruptions may result in mutations or other deleterious effects.

Radionuclides are characterized by the type and energy level of the radiation emitted. Radionuclides contained in WAG 1 soils produce external radiation exposures principally through the production of beta, gamma, and alpha radiation.

The general health effects of radiation can be divided into stochastic and nonstochastic effects (i.e., those health effects not related to threshold dose and those related to threshold dose). Developing cancer from exposure to any amount of radiation is a stochastic effect. Examples of nonstochastic effects include acute radiation syndrome and cataract formation, both of which occur only at high levels of exposures.

Radiation can damage cells in different ways. First, radiation can cause damage to the strands of genetic material, DNA, in a cell. The cell may not be able to recover from this type of damage, or the cell may live on in a functionally abnormal condition. If the abnormally functioning cell divides and reproduces, a tumor or mutation in the tissue may develop. The rapidly dividing cells that line the intestines and the stomach and the cells that make blood in the bone marrow are very sensitive to this kind of damage. Organ damage results from the damage caused to the individual cells. This type of damage has been reported with doses of 10 to 500 rem. Acute radiation sickness is seen only after doses of greater than 50 rem. This dose is usually only received by personnel in proximity to serious nuclear accidents. Principal adverse effects associated with exposure to ionizing radiation are carcinogenicity, mutagenicity, and teratogenicity.

When cells damaged by radiation are reproductive cells, genetic damage can occur in the offspring of the person exposed. The developing fetus is especially sensitive to radiation. The type of malformation that may occur is related to the stage of fetal development and the cells that are differentiating at the time of exposure. Radiation damage to children exposed while in the womb is related to the dose that the pregnant mother received. Mental retardation is another possible effect of fetal radiation exposure.

The following subsections provide additional information about the specific radionuclide COPCs at WAG 1.

6.4.3.6.1 Americium-241—Am-241 is produced by the beta decay of Pu-241. This isotope has been distributed widely in the environment as a result of nuclear weapons fallout. Am-241 decays by alpha emission, which makes the isotope important for internal exposure, whether it is ingested or inhaled. The alpha decay is accompanied by emission of gamma of radiation 60 keV with an abundance of 36%, which is of concern where Am-241 is concentrated, but is not important at environmental levels. The International Committee on Radiological Protection (ICRP) has assigned a value of 5.00E–04 to f_1 for all compounds of americium. For inhalation exposures, the ICRP recommends assigning all compounds of americium to inhalation Group W. Most (90%) of the americium entering the blood stream is deposited in the liver and the bone, with only a small amount being deposited in human reproductive organs. The biological half-lives in the liver and the bone are 40 and 100 years, respectively. The amount deposited in reproductive organs is considered to remain permanently.

6.4.3.6.2 Cobalt-60—Co-60 is an activation product of the irradiation of stable cobalt or nickel in nuclear reactors or nuclear weapons testing. Co-60 is a major component of normal reactor effluent, and is intensely radioactive, emitting characteristic gamma radiation of 1.2 and 1.3 MeV. Co-60 has a radioactive half-life of 5.27 years, but its resident half-life in the human body is only 9.5 days, which is important in limiting the overall exposure from this radionuclide.

The critical organ for Co-60 exposure is the whole body. The SF, which is expressed per unit intake as exposure, is a function of the route of entry.

6.4.3.6.3 Cesium-134—Cs-134 has a radioactive half-life of 2.07 years. It behaves similarly to Cs-137.

6.4.3.6.4 Cesium-137—Cs-137 is a fission product produced in nuclear reactors and in nuclear weapons detonations. Cs-137 is rapidly absorbed into the bloodstream and distributes throughout the active tissues of the body. Metabolically, Cs-137 behaves as an analog of potassium. Its distribution throughout the body and energetic beta and gamma radiation from its daughter, Ba-137m, result in essentially whole-body irradiation (Amdur, Doull, and Klassen 1991). The radioactive half-life of Cs-137 is 30 years. Its biological half-life in adults is 50 to 150 days, in children, 44 days. Cs-137 exists in secular equilibrium with Ba-137m, which is the major contributor to the dose received from a 0.662 MeV gamma ray. The critical organ for Cs-137 exposure is the whole body.

6.4.3.6.5 Curium-242—Cm-242 is an alpha-emitting radionuclide. In the body it behaves similarly to plutonium. Approximately 50% all the curium isotopes that enters the blood are retained in the bone and 30% in the liver with retention times of 20 to 50 years. Studies have shown that after inhalation, less than 1% of the alveollar-deposited curium oxide is found in the thoracic lymph nodes of dogs at greater than 1 year post exposure. Via injection, the initial fraction of Cm-242 in rat liver was three times higher than that of Pu-239, but Cm-242 was also cleared from the liver quickly. Inhalation can cause lung tumors in rats and dermal adsorption is limited (BEIR IV 1988).

Cm-242 has a radioactive half-life of approximately 163 days.

6.4.3.6.6 Curium-244—The radioactive half-life of Cm-244 is 18.1 years. It behaves similarly to Cm-242.

6.4.3.6.7 Europium-154—The radioactive half-life of Eu-154 is 8.59 years. It also behaves similarly to Cs-137.

6.4.3.6.8 Europium-155—The radioactive half-life of Eu-155 is 4.71 years. It also behaves similarly to Cs-137.

6.4.3.6.9 Niobium-94—The radioactive half-life of Nb-94 is approximately 2E+04 years. It decays by emitting beta/gamma radiation and behaves in the body similarly to Sr-90.

6.4.3.6.10 Neptunium-237—Np-237 has a radioactive half-life of 2.1E+06 years, and decays by emitting alpha radiation. Np-237 is produced through the decay of Pu-241 and Am-241, and behaves in the body similarly to the plutonium isotopes.

6.4.3.6.11 Plutonium-239—The main source of plutonium in the environment is from nuclear-weapons testing, with smaller contributions from accidents and space power systems burnup in the atmosphere. U.S. soil contains an estimated 5E-02 pCi/g of plutonium in the top 5 cm (4 in.).

Pu-239 has a radioactive half-life of 2.41E+04 years. Pu-239 decays by alpha emission, thus its mode of decay is accompanied by emission of x- and gamma radiation that are low energy and do not contribute significantly to radiation dose at environmental levels.

Plutonium, which is absorbed into the blood stream, is deposited mainly in the liver and bones [Agency for Toxic Substance Disease Registry (ATSDR 1989)]. For dosimetric purposes, all isotopes of plutonium are assumed to be uniformly distributed over all bone surfaces at all times following deposition.

6.4.3.6.12 Strontium-90—Strontium is an alkaline earth element and is, therefore, chemically similar to calcium and barium. Strontium follows calcium through food chains from environment to organism, but some degree of discrimination exists against strontium (Kirchman et al. 1993). Sr-90 is formed during nuclear fission. Sr-90 has a radioactive half-life of 29.1 years and decays by beta emission. Sr-90's daughter radionuclide, Y-90, also is radioactive. Y-90 has a radioactive half-life of 64 hours and decays by beta emission to the stable isotope Zr-90. Because Sr-90 is produced in the fuel of nuclear reactors, some Sr-90 in the environment at the INEEL may be from reactors operations or from waste transported onto the site. Small amounts of Sr-90 produced in reactor fuel may reach the coolant through defects in the fuel cladding. In coolant purification or following coolant leakage, Sr-90 may reach the gaseous or liquid or both effluent streams and, in controlled amounts, be released to the environment.

In terrestrial ecosystems, 60 to 80% of Sr-90 from fallout is retained in the upper 5 cm (4 in.) of undisturbed soil (Horne 1978). The rate of movement of Sr-90 in soil typically is slow and depends on soil type; low cation exchange capacity, and rapid water movement, and high electrolyte concentrations increase migration rates. Plants acquire Sr-90 by root uptake and direct deposition.

Sr-90 has a radioactive half-life of 29.1 years, and a biological half-life of 49 years in bone and 36 years in the whole body. The target organ for Sr-90 exposure is the bone marrow. Biologically, it

mimics calcium. Sr-90 emits high energy beta particles and its effective dose is high because it concentrates in a relatively small area of the body.

Sr-90 exists its secular equilibrium with its daughter, Y-90, which decays and also contributes to the dose received by the target organism.

Chronic ingestion of radio-strontium in laboratory animals produces a high incidence of myeloproliferative disease, including frank leukemia, at the highest levels of intake. Chronic intake of radio-strontium results in only a few bone tumors in swine while a high incidence was observed in dogs. Researchers have found that radio-strontium data do not fit the linear dose relationship over a wide dose range and that at low levels the data were fit well by practical threshold or sigmoid response relationships.

6.4.3.6.13 Tritium—Tritium (H-3) is a naturally occurring isotope of hydrogen that has also been produced anthropogenically for use in the U.S. weapons program and for various scientific purposes. The radioactive half-life of tritium is approximately 12.3 years, and it decays by producing low energy beta radiation. Because it is an isotope of hydrogen, tritium is most often associated with sources of water in nature. Tritium passes through the body at the same rate as water and causes damage to tissues through ionization caused by its beta radiation.

6.4.3.6.14 Uranium-234, -235, -238—Natural uranium contains three isotopes: U-234, U-235, and U-238. The percent abundance of each isotope in natural uranium is, respectively, 0.006%, 0.72%, and 99.27% (ATSDR 1990). Uranium can be found in the earth's crust at an average concentration of 2 ppm. The ambient air concentration of uranium in the United States ranges from 0.3 to 0.011 fCi/m^3 (1fCi = 10^{-3} pCi). The concentration in drinking water ranges from 0.07 to 653 pCi/L with a median value of 0.1 to 0.2 pCi/L. The average daily intake of uranium has been established to be 0.007 pCi/day from air (0.01 mg/day), 0.7 to 1 pCi/day from food (1 to 1.4 mg/day), and 0.6 to 2.0 pCi/day (0.83 to 2.78 mg/day) from drinking water.

In natural uranium, the radioactivity from U-238 accounts for about half the total radioactivity, and the radiation from U-234 and U-235 accounts for the other half. Uranium emits primarily alpha radiation that is unable to penetrate skin, but can travel short distances in the body if uranium is inhaled or ingested. Natural uranium emits very small amounts of gamma radiation that can penetrate the skin; therefore, little, if any, danger exists from this type of radiation from uranium (ATSDR 1990). Moreover, no human or animal studies have definitively linked inhalation of or oral exposure to natural uranium to the development of cancer.

For noncancer health risks associated with uranium, exposure to natural concentrations of uranium in food, water, air, and soil does not appear to have any toxic effects. Animals that have had oral ingestion of, inhalation of, or dermal exposure to large amounts of uranium have developed damage to the kidney tubules, but other systems were not affected.

The only significant systemic health risk in humans from exposure to nonenriched uranium is potential damage to the kidneys. However, epidemiological studies have not noted an increase in deaths from urogenital or renal diseases, and intravenous studies have failed to identify significant damage to human kidneys following exposure to uranium (ATSDR 1990). Overall, studies in animals and humans also indicate that exposure to uranium is unlikely to produce immunological or neurological effects. Although the data are conflicting, animal studies indicate that exposure to uranium may affect fetal weight and skeletal development in animals, and may possibly alter the ratio of male to female live births in areas where people have excessive exposure to uranium (ATSDR 1990). With the exception of soluble salts, no

oral or inhalation RfDs are available for uranium on IRIS or HEAST, nor has ATSDR established minimum risk levels for different environmental media (EPA 1994; ATSDR 1990).

6.5 Risk Characterization

Risk characterization involves estimating the magnitude of the potential adverse human health effects from released COPCs. Specifically, risk characterization involves combining the results of the exposure and toxicity assessments to provide numerical estimates of health risk. These estimates are either comparisons of exposure levels with appropriate RfDs or estimates of the lifetime cancer risk with a given intake.

6.5.1 Generalized Approach

To quantify human health risks, contaminant intakes are calculated for each COPC by way of each applicable exposure route (see Section 5.3 and Tables B-58 through B-63). As discussed in Section 6.3, these contaminant intakes are based on measured concentration estimates at each retained release site. To determine human health risks, the contaminant specific intakes are compared to the applicable chemical-specific toxicity data discussed in Section 6.4. The following subsections discuss the equations that are used to calculate risks for each retained site.

6.5.1.1 Carcinogenic Health Effects. The following calculations are used to obtain numerical estimates, (i.e., unitless probability) of lifetime cancer risks:

$$Risk = Intake \times SF \tag{6-19}$$

where

Risk	=	potential lifetime cancer risk (unitless)
SF	=	slope factor, for chemicals (mg/kg/day)-1, or radionuclides (pCi)-1
Intake	=	chemical intake (mg/kg/day), or radionuclide intake (pCi).

The linear low-dose equation shown in Equation (6-15) is valid at low risk levels (i.e., below estimated risks of 1E–02). In accordance with *EPAs RAGS* (EPA 1989a), risks that are greater than 1E-02 are calculated using the following one-hit equation:

$$Risk = 1 - exp \ (-Intake \times SF) \tag{6-20}$$

where

Risk	=	potential lifetime cancer risk (unitless)
SF	=	slope factor, for chemicals (mg/kg/day)-1, or radionuclides (pCi)-1
Intake	=	chemical intake (mg/kg/day), or radionuclide intake (pCi).

To develop a total risk estimate for a given release site, the contaminant risks are summed for each COPC at the site.

$$Risk_T = \sum Risk_i \qquad (6\text{-}21)$$

where

$$Risk_T \quad = \quad \text{total cancer risk, expressed as a unitless probability}$$

$$Risk_i \quad = \quad \text{risk estimate for the } i\text{th contaminant.}$$

Similarly, the risk values for each exposure route are summed to obtain the total cancer risk for each potential carcinogen.

6.5.1.2 Noncarcinogenic Effects.
Health risks associated with exposure to individual noncarcinogenic compounds are evaluated by calculating HQs. The HQ is the ratio of the intake rate to the RfD, as follows:

$$HQ = Intake/RfD \qquad (6\text{-}22)$$

where

$$HQ \quad = \quad \text{noncancer hazard quotient (unitless)}$$

$$Intake \quad = \quad \text{chemical intake (mg/kg/day)}$$

$$RfD \quad = \quad \text{reference dose (mg/kg/day).}$$

HIs are calculated by summing the HQs for each chemical across all exposure routes. If the HI for any COPC exceeds 1.0 there may be concern for potential health effects from exposure to the COPC. The HI is calculated using the following equation:

$$HI = \sum \frac{Intake_i}{RfD_i} \qquad (6\text{-}23)$$

where

$$HI \quad = \quad \text{hazard index (unitless)}$$

$$Intake_i \quad = \quad \text{exposure level (intake) for the } i\text{th toxicant (mg/kg/day)}$$

$$RfD_i \quad = \quad \text{reference dose for the } i\text{th toxicant (mg/kg/day).}$$

In the above equation, intake and RfD are expressed in the same units and represent the same exposure time period.

6.5.2 Estimates of Human Health Risk

Estimates of WAG 1 human health risks during each evaluated time period (see Section 6.3 for a discussion of exposure time periods) are presented in Tables B-65 through B-70 and Figures 6-4 through 6-8. For each time period, carcinogenic risks and noncarcinogenic HIs are shown in separate tables and figures.

As discussed in Section 6.3, risk and HI estimates for the air and groundwater pathway exposure routes (i.e., inhalation of fugitive dust, inhalation of volatiles, ingestion of groundwater, dermal absorption of groundwater, and inhalation of water vapor from indoor water use) are calculated in a cumulative manner. As a result, for a given air or groundwater pathway exposure route within a given time period, the risk estimate for the exposure route is the same at every release site.

Risk and HQ estimates for ingestion of groundwater containing maximum predicted COPC concentrations are shown in Tables B-71 and B-72. These risk estimates are presented separately because the maximum predicted COPC concentrations may occur beyond the exposure time periods evaluated in the BRA.
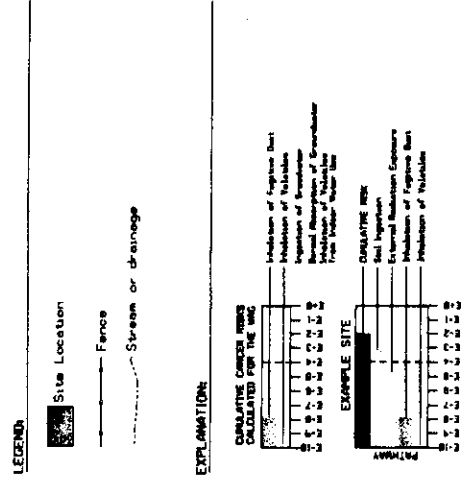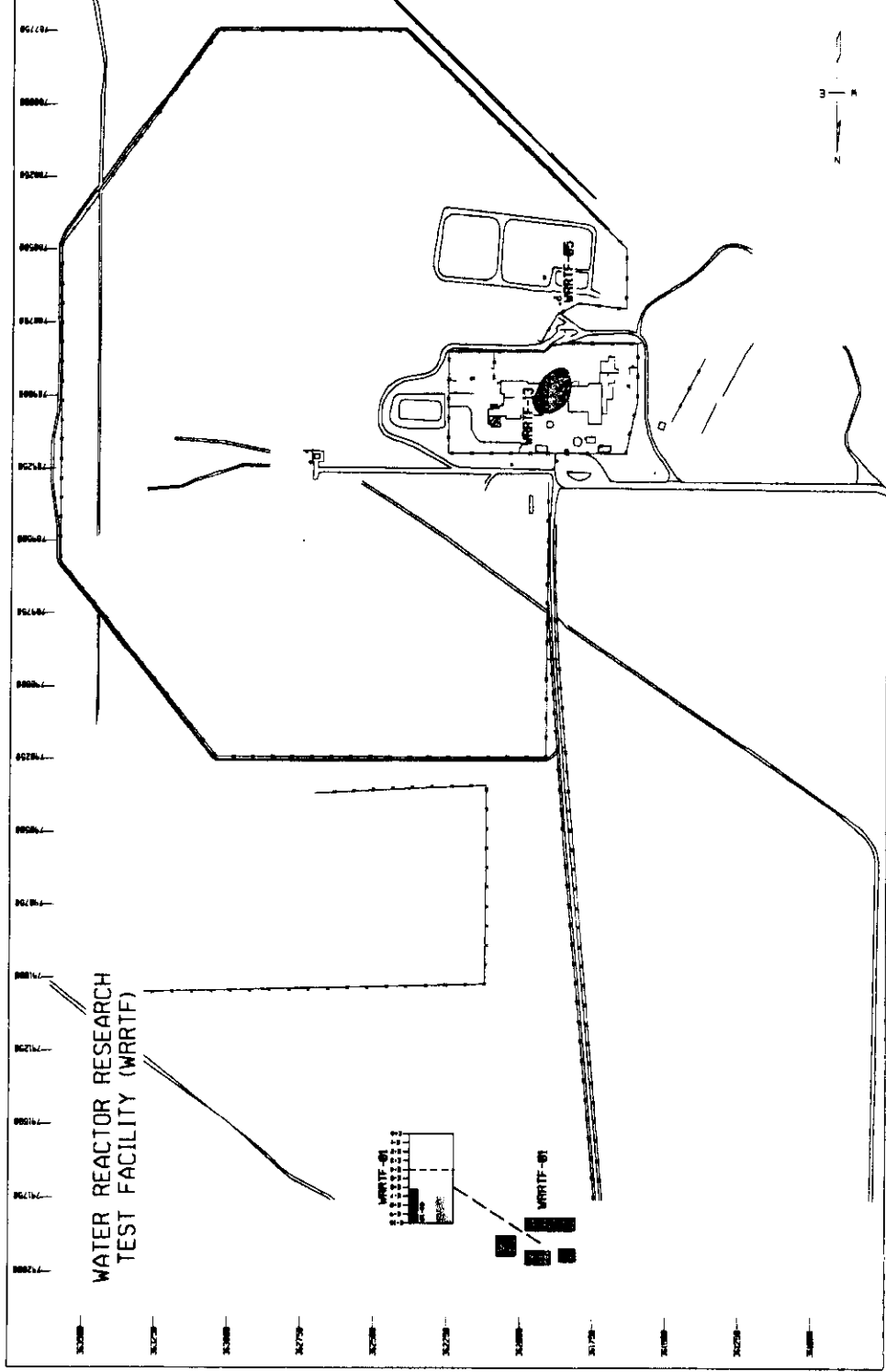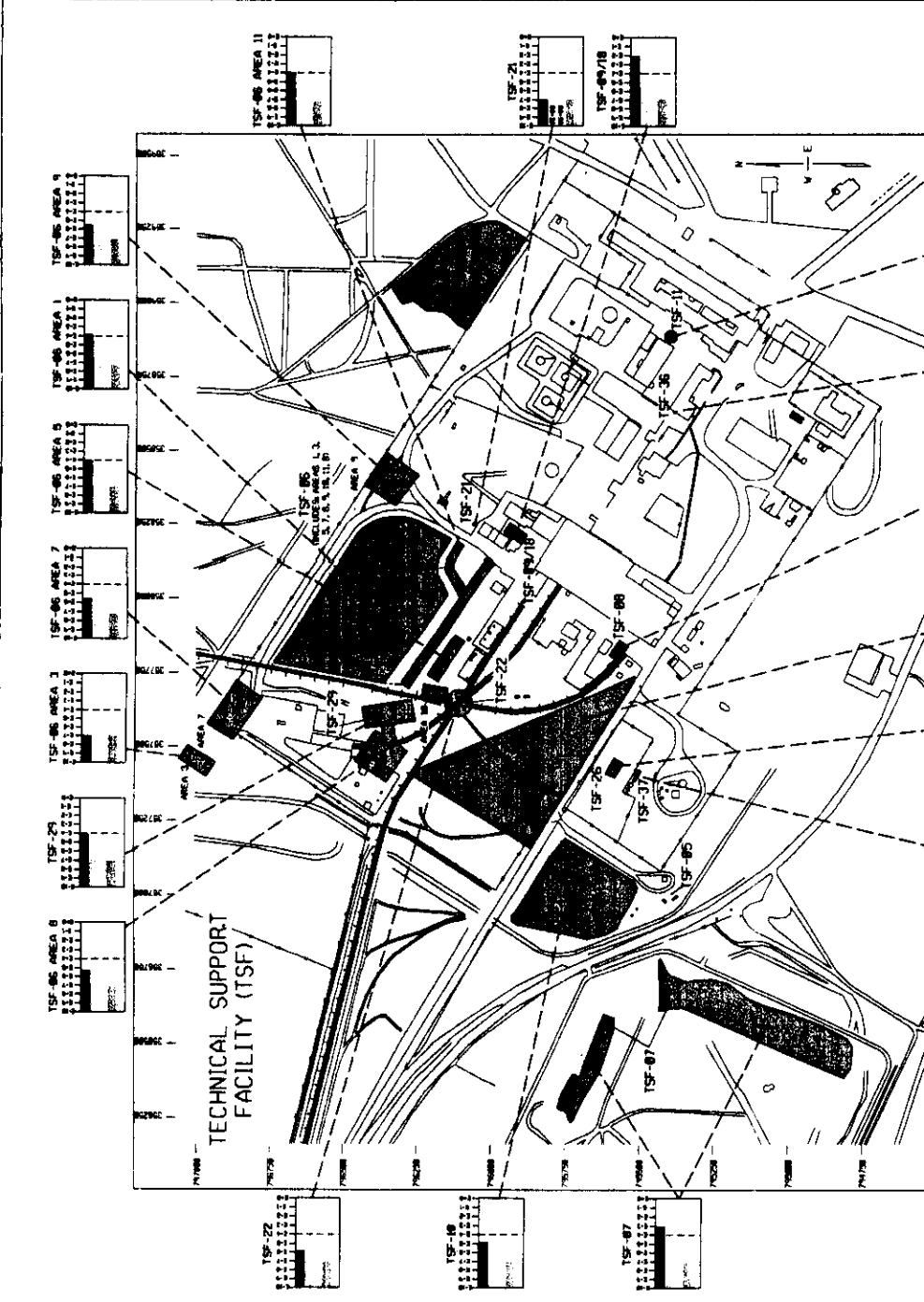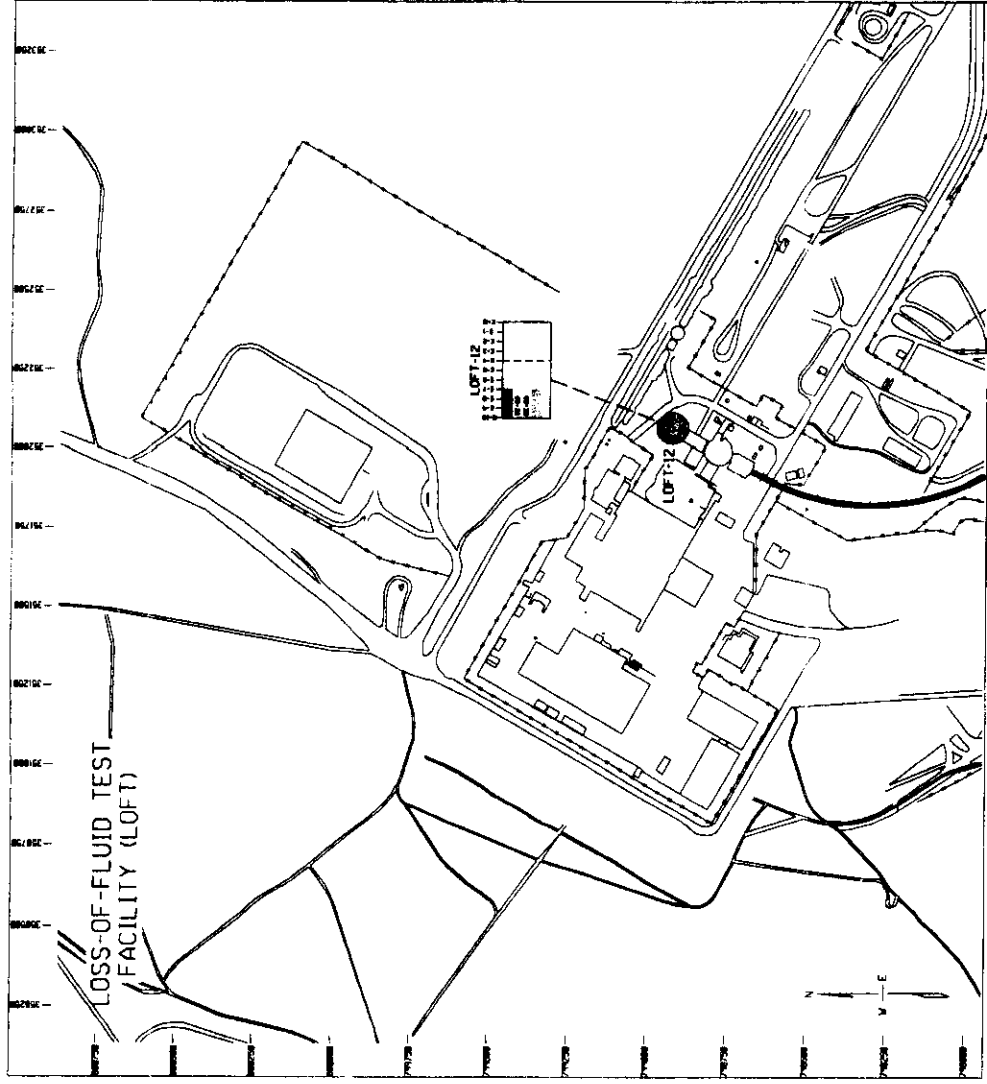
6.6 Uncertainty Analysis

The risk assessment results presented in this BRA are very dependent on the methodologies described in Section 6.3. These analysis methods were developed over a period of several years by INEEL risk management and risk assessment professionals to provide realistic, and yet conservative, estimates of human health risks at WAG 1. Nonetheless, if different risk assessment methods had been used, the BRA would likely have produced different risk assessment results. To ensure that the risk estimates are conservative, health protective assumptions that tend to envelope the plausible upper limits of human health risks are used throughout the BRA. Therefore, risk estimates that may be calculated by other risk assessment methods are not likely to be significantly higher than the estimates presented in Section 6.5.

The BRA results in Section 6.5 are useful for evaluating which WAG 1 release sites require remediation because the results are calculated in a consistent manner. This consistency allows for direct comparison of the risk assessment results for a given release site with the results for every other site included in the evaluation. Changes in a given assumption used in the evaluation would, in general, produce similar changes in the risk results for all of the release sites evaluated. As described in the remainder of this section, the BRA results include inherent uncertainty, but despite this uncertainty, the consistency of the analysis makes the results useful for making remediation decisions.
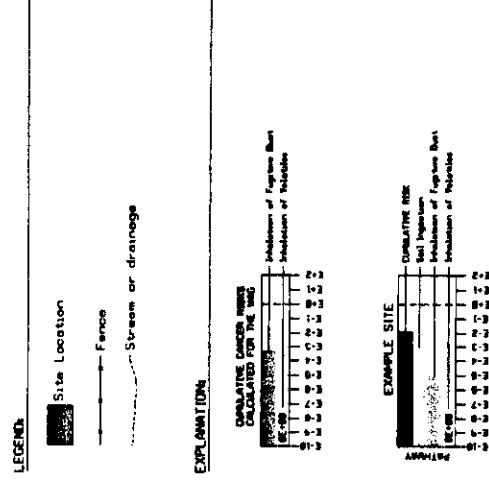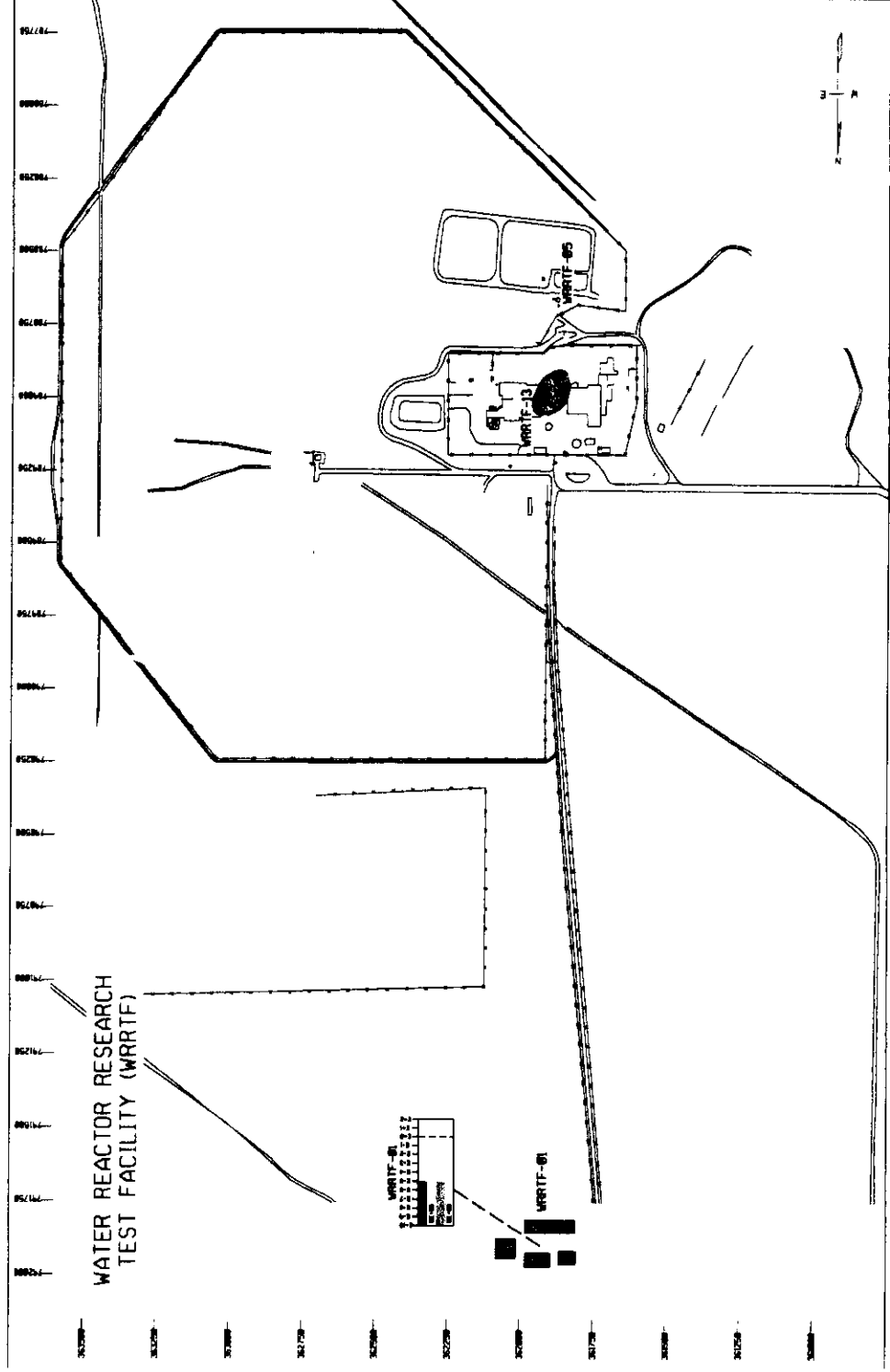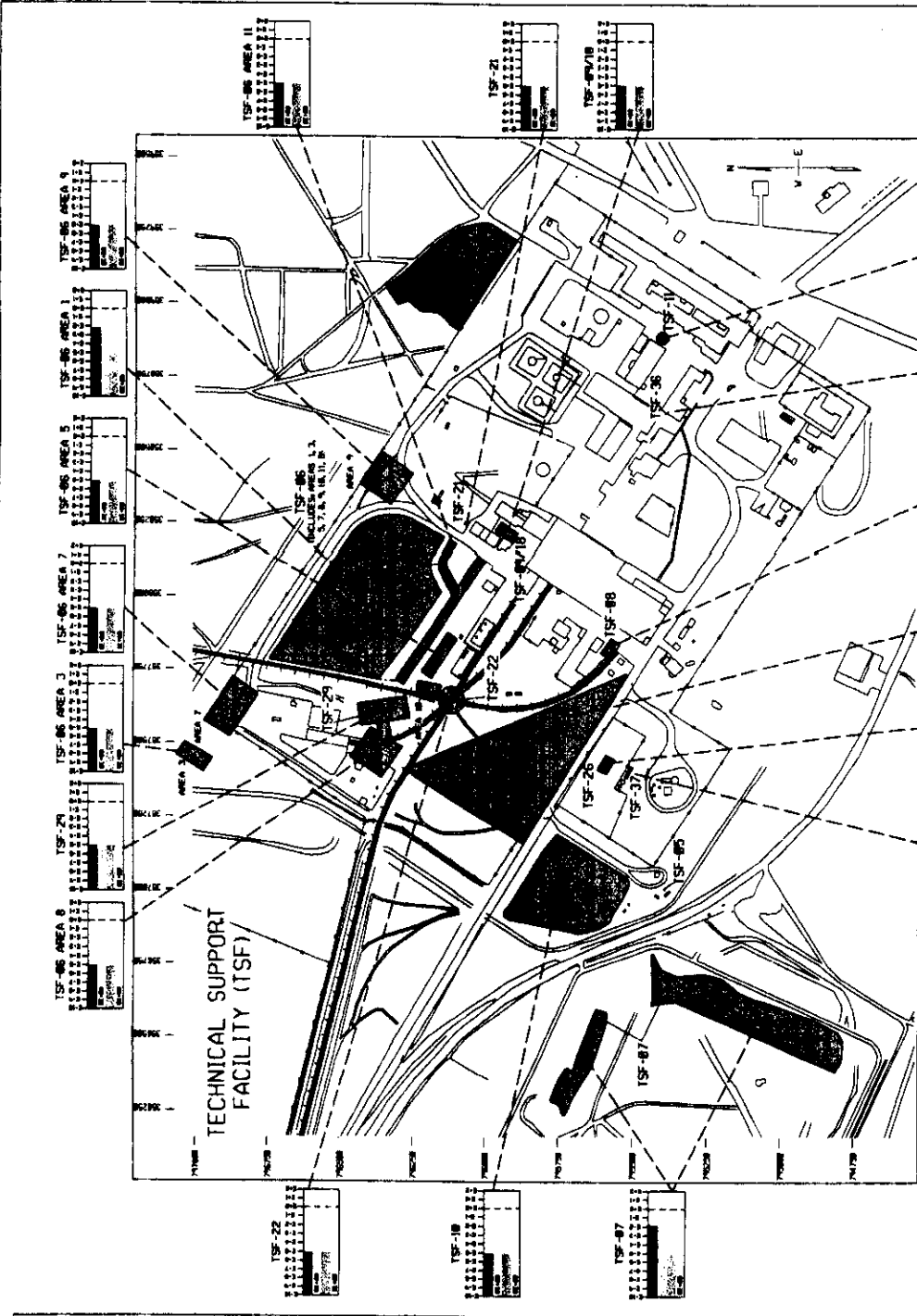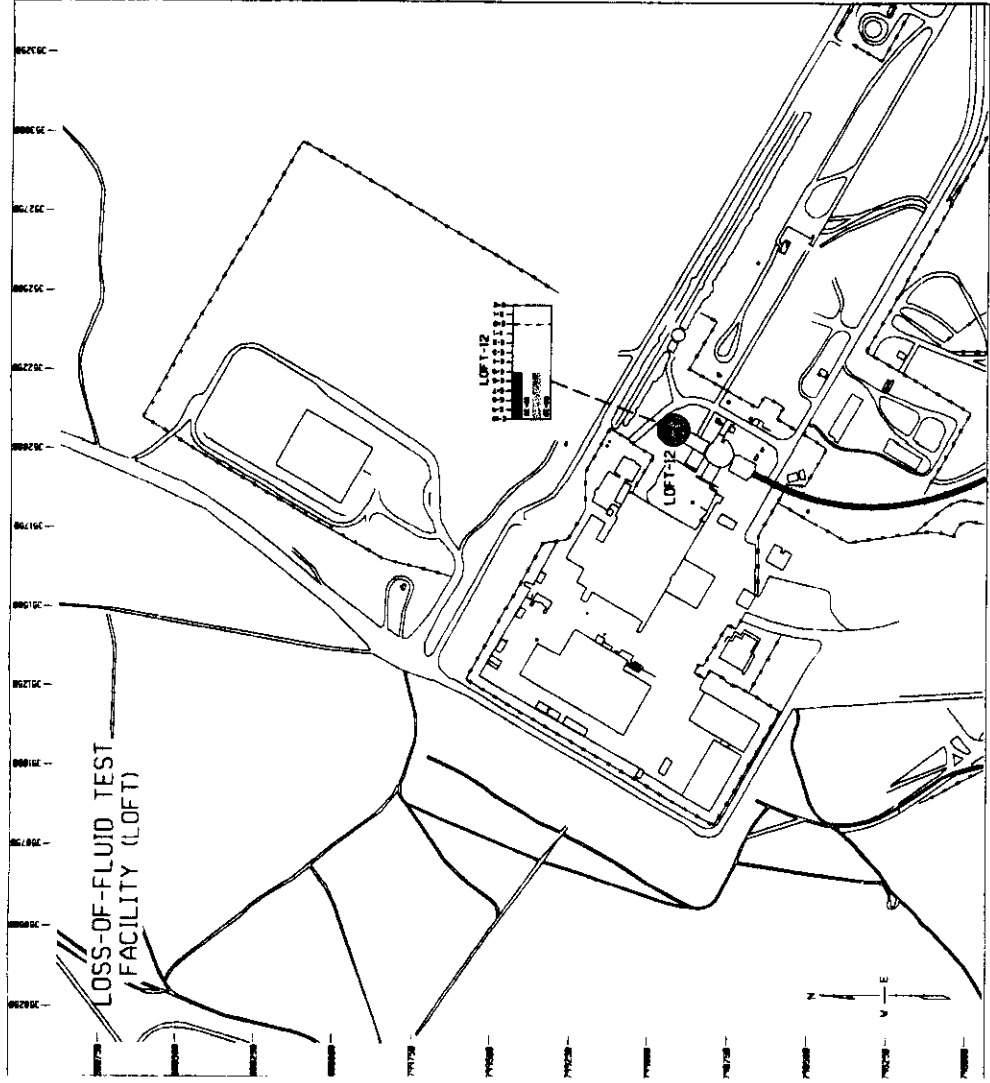
Uncertainty in this BRA is produced by uncertainty factors in the following four stages of analysis:
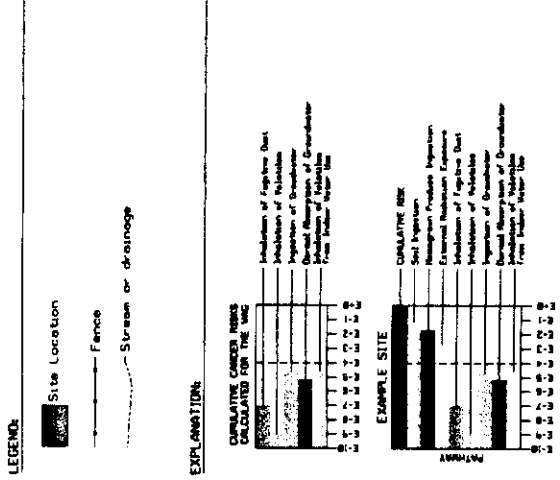
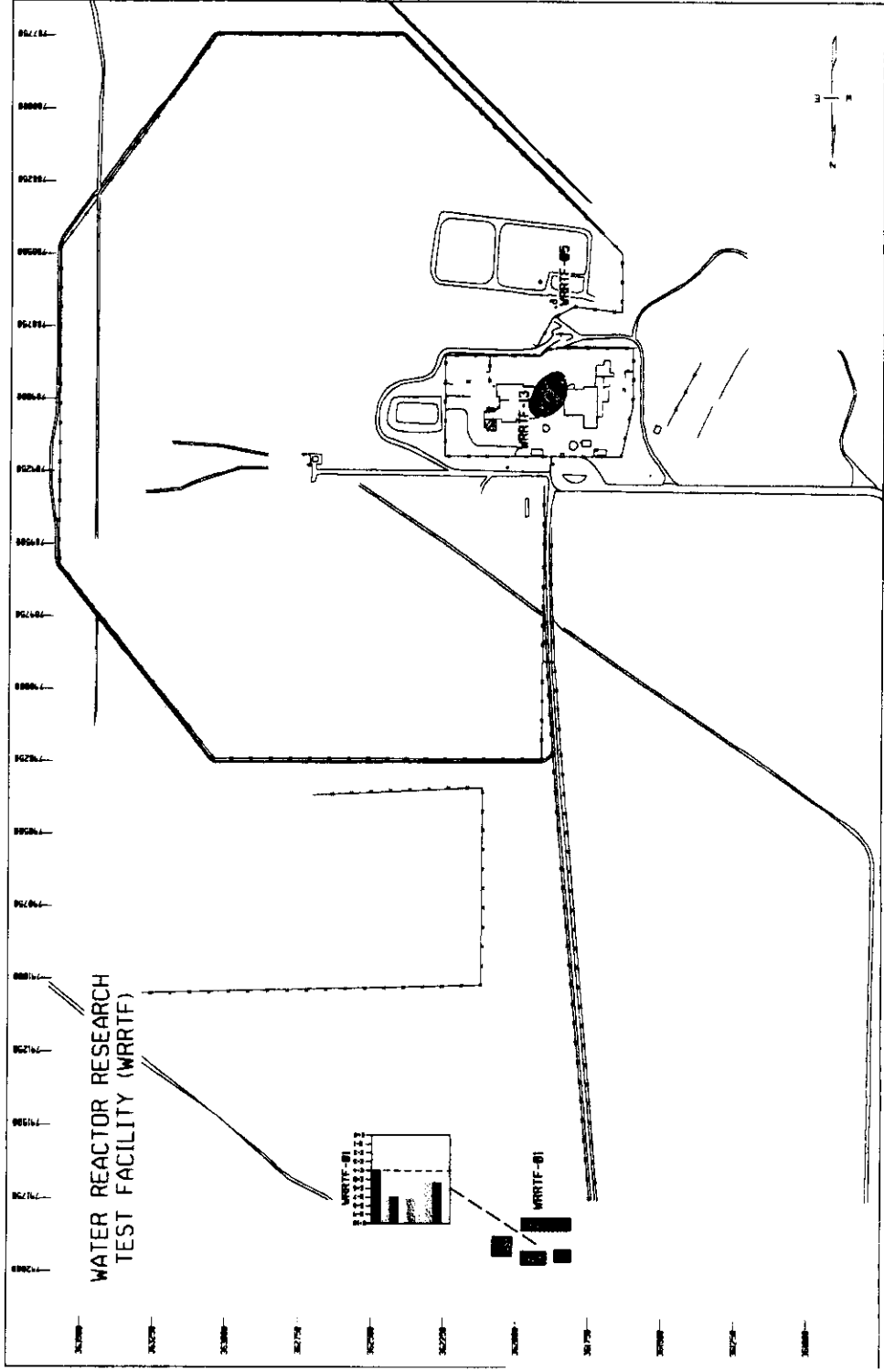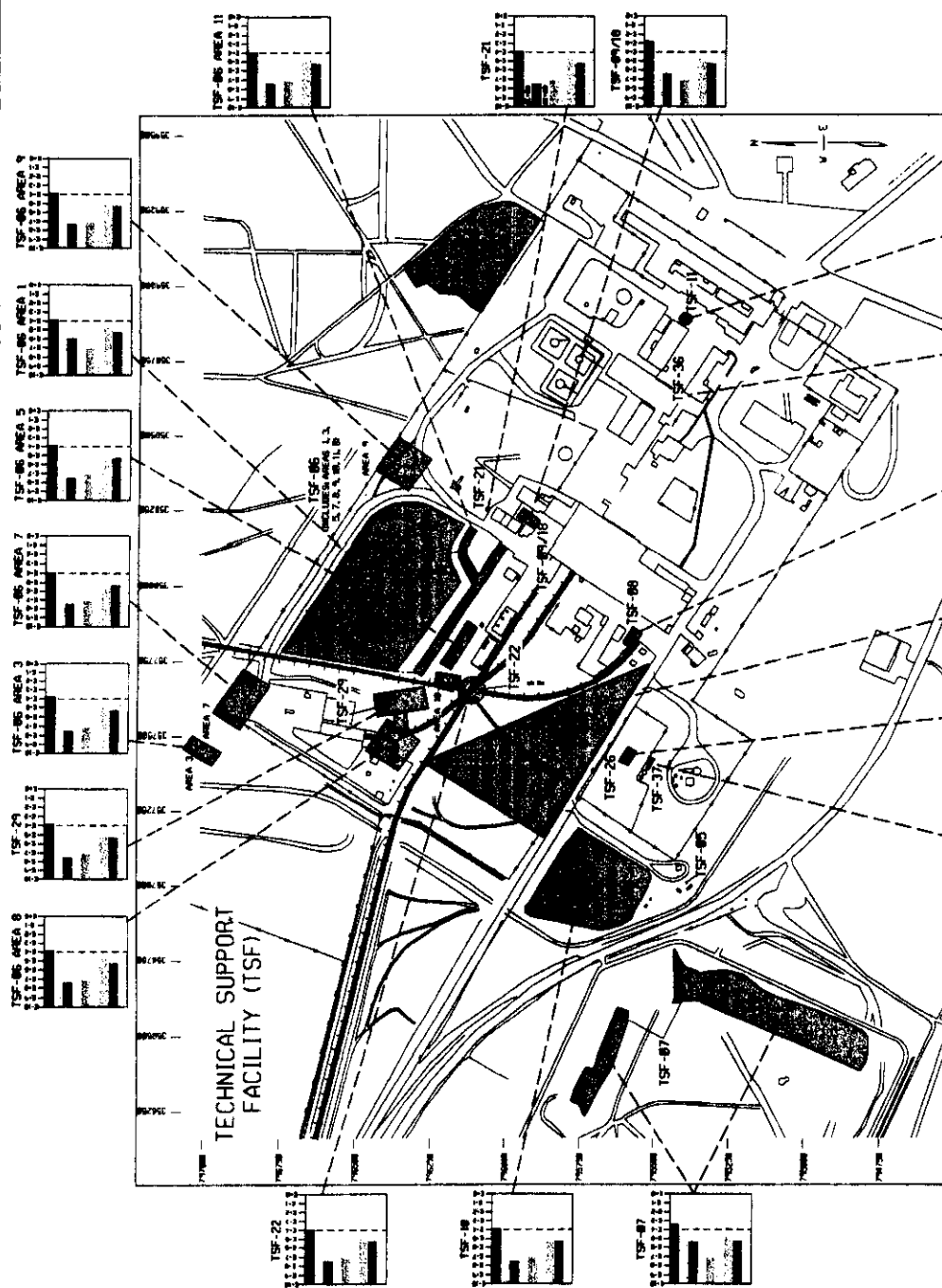1.   Data collection and evaluation

2.   Exposure assessment

3.   Toxicity assessment

4.   Risk characterization.

TECHNICAL SUPPORT
FACILITY (TSF)

LOSS-OF-FLUID TEST
FACILITY (LOFT)

WATER REACTOR RESEARCH
TEST FACILITY (WRRTF)

LEGEND:
Site Location
Fence
Stream or drainage

SCALE IN FEET

TECHNICAL SUPPORT
FACILITY (TSF)

LOSS-OF-FLUID TEST
FACILITY (LOFT)

WATER REACTOR RESEARCH
TEST FACILITY (WRRTF)

LEGEND:

■ Site Location
— Fence
--- Stream or drainage

SCALE IN FEET

IDAHO NATIONAL ENGINEERING LABORATORIES
OU 3-13 HUMAN HEALTH EVALUATION
RI/BRA
CUMULATIVE RISK ASSESSMENT

Carcinogenic Risk for
Worker at 100 Years

6-5

TECHNICAL SUPPORT FACILITY (TSF)

LOSS-OF-FLUID TEST FACILITY (LOFT)

WATER REACTOR RESEARCH TEST FACILITY (WRRTF)

TECHNICAL SUPPORT FACILITY (TSF)

LOSS-OF-FLUID TEST FACILITY (LOFT)

WATER REACTOR RESEARCH TEST FACILITY (WRRTF)

TECHNICAL SUPPORT FACILITY (TSF)

LOSS-OF-FLUID TEST FACILITY (LOFT)

WATER REACTOR RESEARCH TEST FACILITY (WRRTF)

IDAHO NATIONAL ENGINEERING LABORATORIES
OU1-10 RI/BRA HUMAN HEALTH EVALUATION
CUMULATIVE RISK ASSESSMENT

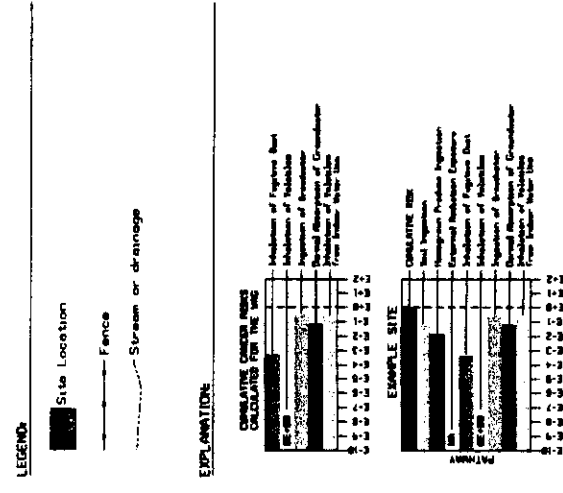Non-Carcinogenic Hazard Indices
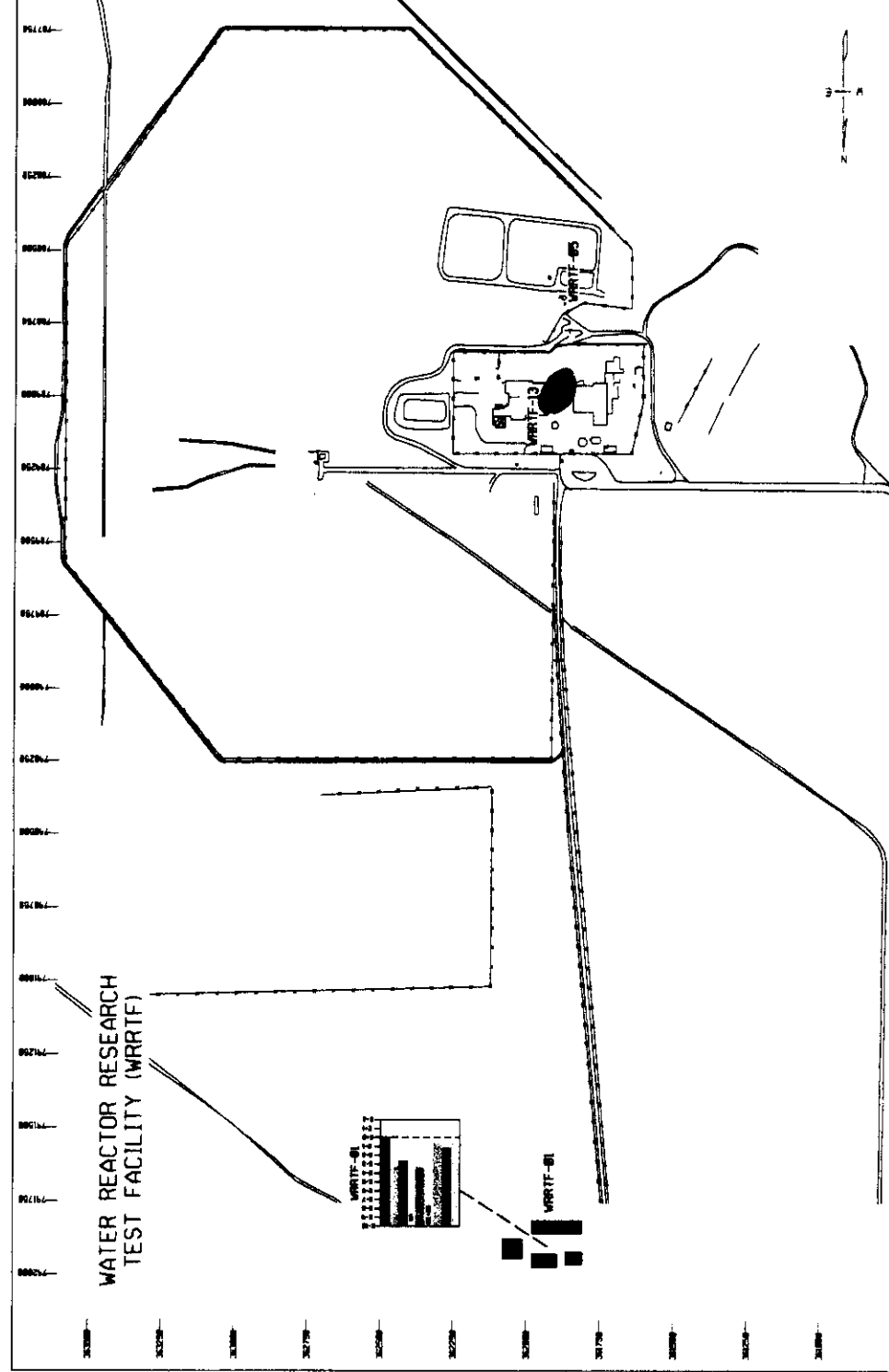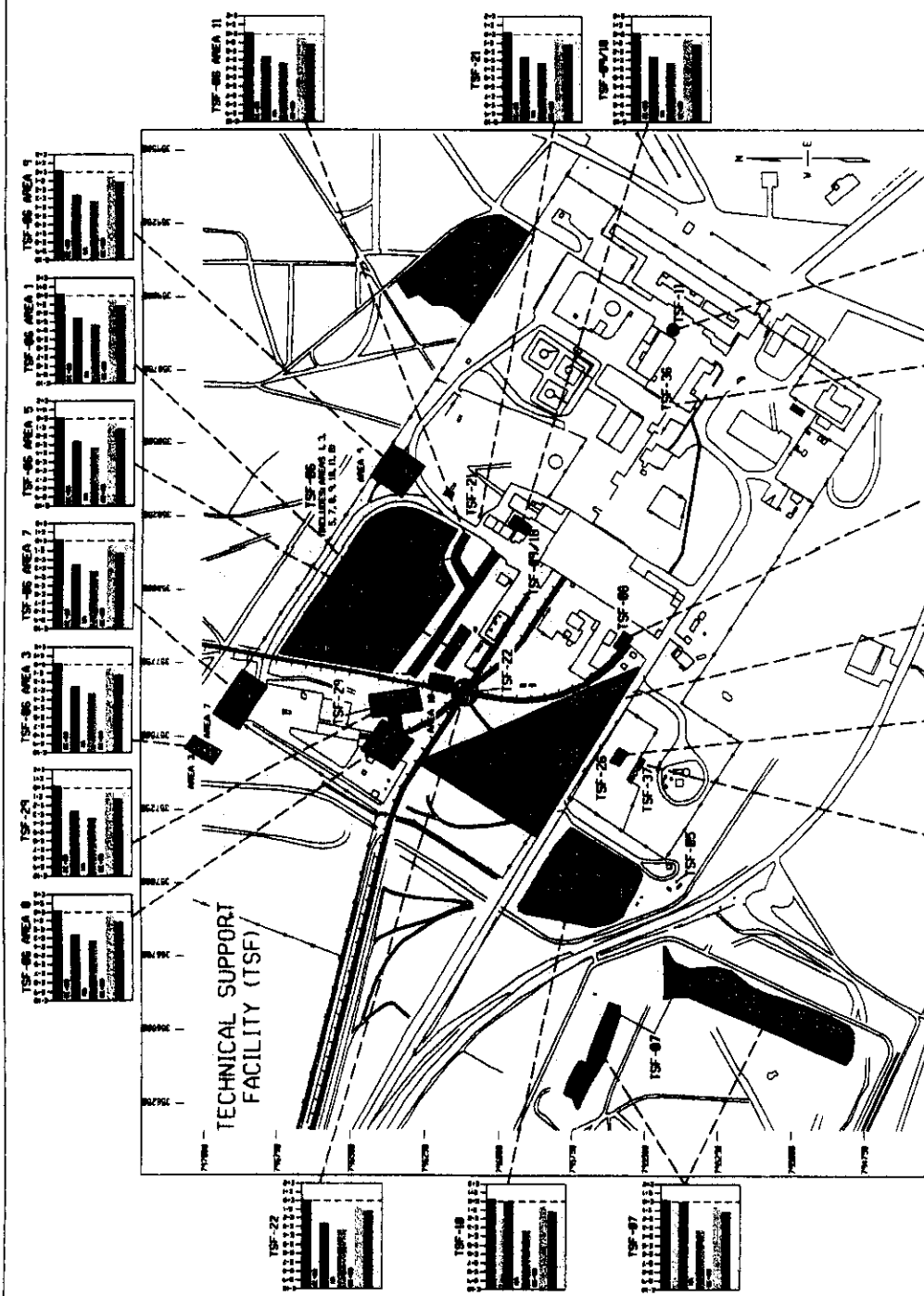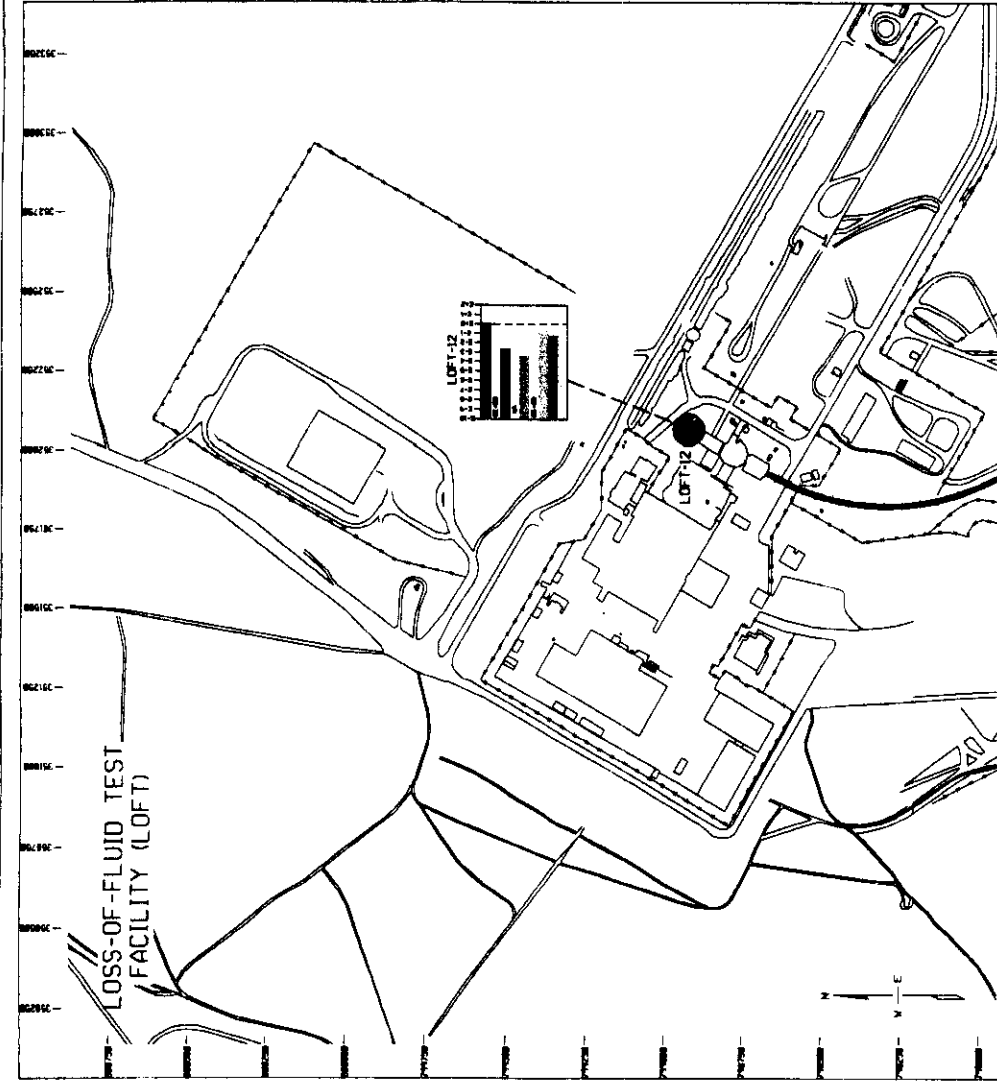For Resident at 100 Years

6-8

The following subsections discuss each of these four stages in more detail, and Section 6.6.5 presents a discussion of risks caused by potential future releases from co-located facilities at WAG 1.

6.6.1 Data Collection and Evaluation Uncertainties

Uncertainties associated with data collection and evaluation are produced by variability in observed concentrations caused by sampling design and implementation, laboratory analysis methods, seasonality, contaminant level variation, and natural concentration variation. Optimizing the usability of sampling data involves quantifying these uncertainties.

The effect of uncertainty introduced from sample collection and analysis is reduced by basing risk estimates on the 95% UCL of the mean for the WAG 1 COPC concentration estimates. The resulting concentration estimates, used to estimate intakes, are an upper bound estimate of the concentrations observed at the retained sites. This approach is health protective and accounts for the uncertainty introduced by sampling, analysis, seasonality, and natural variation.

A major assumption included in the BRA analysis is that all significant sources of contamination at WAG 1 have been identified and sampled. If a source of contamination has not been identified and sampled, the risks from the contamination are not included in the BRA.

One of the first steps in the BRA was a screening of release sites and contaminants (see Section 6.2). The purpose of this screening activity was to help focus the BRA on sites and contaminants that are likely to produce adverse human health effects. The screening process was designed to be conservative so that all sites and contaminants that have a reasonable potential for causing adverse human health effects would pass the screening, and therefore would be evaluated in the BRA. If in fact the screening process was not conservative enough, and sites or contaminants that could cause adverse human health effects were inappropriately screened out, then the BRA risk results presented in Section 6.5 would be underestimated. A contamination source would have to be small to be inappropriately screened, so any underestimation of risk would be slight if a site or contaminant were inappropriately screened.

Two WAG 1 sites, TSF-06: Area 10 (the buried reactor vessel site) and IET-04 (the buried IET stack rubble site) include known radionuclide contamination that was not evaluated in the BRA. The contamination at these sites is fixed to the material that is buried at the sites. In the case of TSF-06: Area 10, the contamination is fixed to the surfaces of a reactor vessel that is buried inside a vault located greater than 3 m (10 ft) belowground surface (bgs), and in the case of IET-04, the contamination is fixed to the surfaces of stack rubble that is also buried greater than 3 m (10 ft) bgs. The contamination at these two sites is not expected to migrate away from the buried material, so the contamination is not expected to cause exposures to humans or ecological receptors at the sites. As a result, risks for the sites are not calculated in Sections 6.5 or 7.

Tentatively identified compounds (TICs) were detected at several of the WAG 1 release sites. These compounds were not included in the BRA risk calculations. In accordance with EPA risk assessment guidance (EPA 1989), the TICs were omitted because they were not detected frequently and because the compounds are not expected to have been released.

Two other WAG 1 release sites, TSF-09/-18 (the V-Tanks), and TSF-26 (the PM-2A Tanks), include highly contaminated liquid and sludge wastes that were not included in the BRA. The risks reported in Section 6.5 and 7 for these two sites are based exclusively on contaminant concentrations detected in the soils surrounding the tanks. There is currently no evidence that the tanks at either of these

two sites have leaked, but if a leak ever did occur, the risks at the sites would likely become much more severe. The wastes inside the tanks were excluded from evaluation in the BRA because the primary goal of the BRA is to evaluate risks from past releases at WAG 1. Evaluation of a hypothetical future release from the tanks is beyond the scope of the BRA. Nonetheless, removal or stabilization of the wastes contained in these tanks will be an important aspect of any remedial action that may be planned at TSF-09/-18 and TSF-26.

All of the release sites evaluated in the BRA have varying levels of uncertainty associated with the contaminant concentrations evaluated in the BRA. Additionally, all of the evaluated concentrations were estimated using conservative assumptions about the nature and extent of contamination at the various release sites. The concentration term uncertainties and conservative assumptions are summarized in Table 6-4.

Twenty-eight contaminants were screened out of the BRA analysis as a result of the RBC screening. GWSCREEN runs were performed to evaluate the impact of these contaminants on groundwater risks. These GWSCREEN runs predict that none of the contaminants produce maximum groundwater ingestion risks greater than 1E-06 or hazard quotients greater than 1 using the analysis methods described in Section 6 of the BRA. As a result, screening these contaminants does not have any effect on the BRA results.

6.6.2 Exposure Assessment

Uncertainties associated with the exposure assessment are produced by characterizing transport, dispersion, and transformation of COPCs in the environment; establishing exposure settings; and deriving estimates of chronic intake. The initial characterization that defines the exposure setting for a site involves many professional judgments and assumptions. Definition of the physical setting, population characteristics, and selection of the chemicals included in the risk assessment are examples of areas for which a quantitative estimate of uncertainty cannot be achieved because of the inherent reliance on professional judgment. Assumptions and supporting rationale regarding these types of parameters, along with the potential impact on the uncertainty (i.e., overestimation or underestimation of uncertainty), are included in Table 6-4.

An aspect of the risk assessment that tends to exaggerate risk results is the evaluation of contaminants with background concentrations that produce calculated risks in excess of 1E–06. Two examples of this type of contaminant are arsenic and beryllium. Both contaminants are commonly detected in INEEL soils at concentrations that are slightly higher than accepted background concentrations. However, neither contaminant is associated with known waste producing processes at WAG 1, and they both have very high toxicity constants. Therefore, arsenic was not included in the risk assessment for the five sites (TSF-9/18, TSF-10, TSF-26, TSF-27, and TSF-36) and beryllium was not included in the risk assessment for one site (TSF-36). If the source of the detected arsenic and beryllium concentrations is anthropogenic (i.e., in the context of the BRA, produced by operations at the sites), which is unlikely, the risk results for the sites are underestimated.

As discussed in Section 4, the contaminant source terms evaluated in the BRA were calculated by "volume weighting" measured site concentrations. Volume weighting refers to the process of measuring contaminant concentrations at various locations and depths at a given release site, estimating the volume of soil that is represented by a given measurement or group of measurements, and deriving average contaminant concentrations at the site by weighting the measurements with the associated soil volumes. This process produces reasonable estimates of a site's average contaminant concentrations as long as the

Table 6-4. Summary of source term uncertainties and conservative assumptions for the OU 1-10 BRA.

Release Site	Source Term Uncertainties and/or Conservative Assumptions
TSF-11: Clarifier Pits	The maximum concentration of Am-241 (the only retained COPC at the site) is assumed to exist over the entire site area 1.2 by 3.0 m (4 by 10 ft) and from a depth of 2.1 m (7 ft) down to the maximum depth of detected contamination. These conservative contamination nature and extent assumptions probably cause the calculated risks at the site to be overestimated.
WRRTF-01: WRRTF Burn Pits	95% UCL on the mean or maximum contaminant concentrations are assumed to exist in each of the site's burn pits over the maximum detected burn layer thickness. These conservative contamination nature and extent assumptions overestimate the risk at the site. Conservative concentration estimates for each of the four pits were averaged together to develop a single site-wide concentration estimate for each of the site's COPCs. This analysis method does not take any credit for the clean soil that is present in between the burn pits, and it does not account for the possibility of unidentified contamination that may exist between the pits.
TSF-29: TSF Acid Pond	95% UCL on the mean or maximum contaminant concentrations are assumed to exist over the entire surface of the post-1958 pond. This conservative contamination nature and extent assumption probably causes the calculated risks at the site to be overestimated. The depth of contamination at the site is conservatively assumed to be 0.8 m (2.5 ft), even though contamination was only detected in the top 0.2 m (6 in.) of soil at the sites. Insufficient samples were collected at depth to determine a true depth of contamination for each COPC. This nature and extent assumption may overestimate or underestimate risk at the site depending on how the assumption compares to the site's true depth of contamination. Radionuclide hotspots were detected within the post-1958 pond during field surveys at the pond. The sampling boreholes at the pond were not drilled at the locations of these hotspots, so the hotspot contamination was not included in the data used to develop the site's concentration terms. Calculations were performed to estimate the contaminant concentrations at the hotspots based on the field survey readings and assuming that the hotspots only contain Cs-137. The concentration estimates ranged from 0.4 pCi/g to 8 pCi/g. These low levels of contamination are lower than the 16.1 pCi/g of Cs-137 evaluated in the BRA, so the hotspot contamination would not have increased the evaluated concentration term. As a result, the site's calculated risks are expected to be overestimated. Despite the presence of the hotspot contamination. All contamination in the TSF-29 pre-1958 pond was evaluated as part of TSF-06: Area 8.
TSF-06 Area 1: Soil northeast of Turntable	95% UCL on the mean concentrations were assumed to exist for each COPC over the entire site surface area, and a conservative site surface area of 183 by 122 m (600 by 400 ft) was assumed based on limited sampling information. No contamination information with depth is available, but the site potentially contains buried waste released from underground pipes. Since there is a possibility that contamination exists at depth, the site's zone of contamination is conservatively assumed to stretch from the surface down 3 m (10 ft). These conservative nature and extent assumptions are expected to overestimate the risk at the site.

Table 6-4. (continued).

Release Site	Source Term Uncertainties and/or Conservative Assumptions
Area 3: TAN-781 Pond	Only one sample that could be used for the RI/BRA nature and extent evaluation was collected at this site. Am-241 was the only COPC identified by the contaminant screening evaluation at the site, and the maximum Am-241 concentration detected from the one sampling location was assumed to exist over the site's entire surface area. The maximum depth of contamination at the site was assumed to be 0.2 m based on the results from the one sampling location. Risks at the site may be overestimated or underestimated based on whether the site's sampling location is representative or not.
Area 5: Radioactive Soil Berm	Maximum detected contaminant concentrations are assumed to exist throughout the contaminated soil berm. This conservative nature and extent assumption tends to overestimate site risk. Contamination at this site is assumed to be limited to the berm material. No migration of contamination into the soil beneath the berm is assumed. This assumption may result in a slight underestimation of the site's risk if migration of contamination has in fact occurred.
Area 7: Soil Box Storage	Maximum detected contaminant concentrations are assumed to exist over the entire site area, and a conservative site surface area of 61 by 46 m (200 by 150 ft) was assumed based on limited sampling information throughout the contaminated soil berm. These conservative nature and extent assumptions tend to overestimate site risk. The depth of contamination for the site was assumed to be 1.2 m (4 ft) since this is the maximum depth of the only vertical composite sample collected at the site. The site risk may be slightly underestimated if the true depth of contamination over a significant portion of the site is greater than 1.2 m.
Area 8: Cask Storage Pad	This site is composed of two contamination zones that overlap. The first zone is the surface contamination associated with the TSF-06 Cask Storage Pad, and the second zone is the deeper contamination associated with the TSF-29 pre-1958 pond. The risk calculations for the site are based on the maximum detected contaminant concentrations from both zones., and the site's contamination is assumed to exist over the entire surface area of the site. The depth of contamination at the site is conservatively assumed to be 3 m (10 ft) even though the maximum detected depth of contamination at the site is 2.3 m (7.5 ft). This assumption accounts for the limited sampling at depth that was performed at this site. The conservative contamination nature and extent assumptions are expected to overestimate the risk at the site.
Area 9: Northeast Corner Soil Contamination	Maximum detected contaminant concentrations are assumed to exist over the entire site area, and a conservative site surface area of 27 by 8 m (90 by 26 ft) was assumed based on limited sampling information. These nature and extent assumptions are expected to overestimate site risk. The depth of contamination at the site is assumed to be 0.8 m (2.5 ft) although no samples were collected at depth. This assumption could cause a slight underestimation of risk if the true depth of contamination over a significant portion of the site is greater than 0.8 m.
Area 10: Buried Reactor Vessel	Contamination at the site is assumed to be fixed to the site's buried reactor vessel. The BRA does not include any risk calculations for the site because human and ecological receptors are not expected to ever become exposed to the site's contamination.

Table 6-4. (continued).

Release Site	Source Term Uncertainties and/or Conservative Assumptions
Area 11: TSF-06 Ditch	The TSF-06 ditch stretches from the western edge of TSF-09/18 (the V-Tanks site) to the eastern edge of TSF-06: Area 3 (the TAN-781 pond). Maximum detected concentrations of the site's COPCs are assumed to exist over the entire length of the ditch. These nature and extent assumptions are expected to overestimate site risk.
	The depth of contamination at the site is assumed to be 0.8 m (2.5 ft) although no samples were collected at depth. This assumption could cause a slight underestimation of risk if the true depth of contamination over a significant portion of the site is greater than 0.8 m.
Area B: Soil South of Turntable	Maximum detected contaminant concentrations are assumed to exist over the entire site area. This nature and extent assumption is expected to overestimate site risk.
	The depth of contamination at the site is assumed to be 0.8 m (2.5 ft) although no samples were collected at depth. This assumption is expected to overestimate risk at the site because the site's contamination is wind blown.
TSF-09/18: V-Tanks	Maximum and 95% UCL on the mean concentrations of the site's COPCs are assumed to exist over the entire site surface area and down to the maximum depth of detected contamination. These conservative nature and extent assumptions are expected to overestimate site risk.
	Site risks were calculated without accounting for the highly contaminated wastes contained in the site's tanks. There is no evidence that the tanks have leaked, but if a leak ever did occur, the site's risks would likely increase.
	There is a possibility that sampling of the site's soils did not detect all contaminants that have been released at the site. Samples of the tank's contents have been collected but not yet analyzed. If these tank samples reveal contaminants that were not analyzed for in the site's soil samples, the site risk may be underestimated since the contamination contained in the site's soils was released during spills associated with filling the site's tanks.
TSF-10: TSF Drainage Pond	Maximum and 95% UCL on the mean concentrations of the site's COPCs are assumed to exist over the entire site surface area. Varying depths of contamination are assumed for each of the site's COPCs. The assumed depths are based on the maximum depths at which each contaminant was detected at the site. These conservative nature and extent assumptions are expected to overestimate site risk.
TSF-21: IET Valve Pit	Calculated risks at the site are based on the maximum detected COPC concentrations at the site. This assumption overestimates site risk.
	The area of contamination at the site was assumed to include the entire excavated area at the site and the soil between the excavated area and the borehole located 4.6 m (15 ft) from the center of the excavated area. The depth of contamination at the site was assumed to stretch between the bottom of the excavated area [3.4 m (11 ft) bgs] and end at 5.9 m (19.5 ft) bgs. The site risk may be overestimated or underestimated depending on whether the site's true zone of contamination is smaller or larger than the assumed zone.
	Surface exposure route risks (e.g., soil ingestion, inhalation of fugitive dust, etc.) were not calculated because the site's contamination is buried greater than 3 m (10 ft) bgs. This site is assumed to only impact the groundwater pathway exposure routes (e.g., ingestion of groundwater, dermal absorption of groundwater, etc.).

Table 6-4. (continued).

Release Site	Source Term Uncertainties and/or Conservative Assumptions
TSF-26: PM-2A Tanks	This site contains two zones of contamination. The first zone is associated with surface spills and covers an area of 107 by 53 m (350 by 175 ft). The depth of contamination throughout this zone is assumed to be 0.2 m (6 in.). The second zone is associated with deeper spills that occurred while filling the PM-2A Tanks. This zone of contamination is assumed to stretch from the surface down to the maximum depth of detected contamination. These nature and extent assumptions overestimate site risk. In 1995 some contamination was removed from the surface contamination zone and replaced with clean soil. No credit is taken for this clean soil in the BRA because the volume of clean soil is unknown. This omission causes an overestimation of the site risk. Site risks were calculated without accounting for the highly contaminated wastes contained in the site's tanks. There is no evidence that the tanks have leaked, but if a leak ever did occur, the site's risks would likely increase. There is a possibility that sampling of the site's soils did not detect all contaminants that have been released at the site. If the PM-2A tanks contain contaminants that were not analyzed for in the site's soil samples, the site risk may be underestimated since the contamination contained in the site's soils was released during spills associated with filling the site's tanks.
TSF-07: TSF Disposal Pond	The zone of contamination at the site is assumed to cover the entire overflow and main pond surface, and is conservatively assumed to stretch from the surface down to a depth of 3.4 m (11 ft) for metals and radionuclide COPCs and to a depth of 1.5 m (5 ft) for organic contaminants. These nature and extent assumptions overestimate site risk. No hotspots have been detected within the disposal pond, so none were evaluated in the BRA. The site risk may be slightly underestimated if hotspot contamination actually does exist within the pond.
TSF-08: Mercury Spill Site 13B	The zone of contamination at the site is assumed to cover the area that was excavated during the removal action at the site. Mercury contamination is assumed to exist from a depth of 0.8 m (2.5 ft) (the bottom of the site's excavation) down to a depth of 3 m (10 ft), and the site's radionuclide contamination is assumed to exist from 0.8 m (2.5 ft) down to a depth of 1.5 m (5 ft). The site risk may be overestimated or underestimated depending on whether the site's true zone of contamination is smaller or larger than the assumed zone.
TSF-22: Railroad Turntable	Maximum detected contaminant concentrations are assumed to exist over the entire area beneath the turntable. This nature and extent assumption is expected to overestimate site risk. The depth of contamination at the site is assumed to be 0.8 m (2.5 ft) although no samples were collected at depth. This assumption could cause a slight underestimation of risk if the true depth of contamination over a significant portion of the site is greater than 0.8 m.
TSF-36: TAN-603 French Drain	Benzo(a)pyrene, the site's only COPC, was detected at a depth of 2.3 m (7.5 ft) bgs in the center of the french drain excavation. The depth of contamination for the COPC is assumed to extend from 2.3 m (7.5 ft) bgs to a depth of 3 m (10 ft) bgs, and it is assumed to cover an area with a 1.5 m (5 ft) radius from the center of the excavation. The site risk may be overestimated or underestimated depending on whether the site's true zone of contamination is smaller or larger than the assumed zone. The site risk is based on the maximum detected benzo(a)pyrene concentration collected after the french drain was removed. Use of the maximum detected concentration overestimates site risk.

Table 6-4. (continued).

Release Site	Source Term Uncertainties and/or Conservative Assumptions
TSF-37: Contaminated Well Water Spill	The Sr-90 zone of contamination at the site is assumed to include soil between a depth of 0.3 m (1 ft) bgs to a depth of 1.2 m (4 ft) bgs. The H-3 zone of contamination is assumed to cover the soil between a depth of 1.2 m (4 ft) bgs to a depth of 1.5 m (5 ft) bgs. An area with a 1.5 m (5 ft) radius from the contaminated liquid spill location is assumed to be contaminated with both contaminants. The site risk may be overestimated or underestimated depending on whether the site's true zone of contamination is smaller or larger than the assumed zone. Calculated risks at the site are based on maximum detected concentrations within the zones of contamination. Use of maximum concentrations overestimates site risks.
LOFT-12: North Transformer Yard	95% UCL on the mean concentrations of Arolclor-1260 (the only COPC identified at the site) are assumed to exist throughout the sites zone of contamination. This nature and extent assumption overestimates site risk. The depth of contamination at the site is assumed to range from 0.3 m (1 ft) to 1.8 m (6 ft) due to variable depths of remediation. The site risk may be overestimated or underestimated depending on whether the site's true zone of contamination is smaller or larger than the assumed zone.
IET-04: IET Stack Rubble	Contamination at the site is assumed to be fixed to the site's buried stack rubble. The BRA does not include any risk calculations for the site because human and ecological receptors are not expected to ever become exposed to the site's contamination.
WRRTF-13: WRRTF Fuel Leak	The BRA does not contain any risk calculations for this site because none of the site's COPCs have toxicity information available.
TSF-03: TSF Burn Pit	The BRA does not contain any risk calculations for this site because none of the site's COPCs have toxicity information available. 95% UCL on the mean or maximum contaminant concentrations are assumed to exist in the burn pit over the maximum detected burn layer thickness. These conservative contamination nature and extent assumptions overestimate the actual extent of contamination in the pit.

site was thoroughly sampled. If the contamination at a given site was not well defined, volume weighting could produce errors in the site's average concentrations. These errors could either over or under estimate the true average contaminant concentrations at the site, depending on the results of the site's sampling investigation. Details of the sampling investigations evaluated in the RI/FS are discussed in Section 4, and summarized in Table 6-4.

The only contaminant loss mechanism considered in the BRA is radioactive decay. Other loss mechanisms such as leaching, wind erosion, etc., are assumed to be negligible. The reason for this assumption is that environmental sampling has shown that most contaminants do not migrate from most INEEL release sites. As a result of this observation, very few studies have been performed to evaluate these mechanisms, so there is very little site specific information available to estimate the exact effects of these removal mechanisms.

Omitting removal mechanisms other than radioactive decay tends to overestimate risk for all exposure routes because it leads to assuming a given mass of contaminant will cause exposures to multiple exposure routes. For example, leaching is omitted in the soil pathway analysis even though leaching is the mechanism that produces the contamination evaluated in the groundwater pathway analysis. As a result of

the omission, a given mass of contamination can affect both the soil pathway and groundwater pathway risk results. Upper bound infiltration and contaminant leachability assumptions are used in the groundwater pathway analysis to estimate future groundwater contaminant concentrations. Applying these same upper bound assumptions to the soil pathway analysis would likely produce an underestimation of soil pathway risks. To avoid this possibility, leaching is omitted from the soil pathway analysis, so that upper bound risk results are calculated for both the soil pathway and groundwater pathway exposure routes.

One of the purposes of the BRA is to estimate upper bound risks from WAG 1 contaminant releases based on best available site specific information. Omitting removal mechanisms that have not been studied on a site specific basis, and which are likely to produce only small errors in the calculated risk results, is consistent with this objective.

Most COPCs are screened from further analysis under the dermal absorption from soil exposure route for three reasons. First, as discussed above, the dermal absorption from soil exposure route will produce smaller calculated risks than the soil ingestion exposure route for all WAG 1 COPCs; second, the ABS values for most WAG 1 COPCs are not well defined and are, therefore, of limited use in calculating dermal absorption risks; and third, organic contaminants, the class of contaminants that are most likely to produce unacceptable dermal absorption risks, are not widespread at WAG 1.

Omitting a complete evaluation of dermal absorption in the BRA could result in, at most, an underestimation of risk by a factor of two. This situation would occur at a site that contains one or more contaminants that produce dermal absorption risks that are equal to soil ingestion risks, if the contaminants produce the highest risk at the site, and the soil ingestion exposure route with the highest calculated risk for the site. This situation does not occur at any WAG 1 site, so omitting the dermal absorption of soil exposure route from further evaluation is not expected to produce an underestimation of cumulative risk for the WAG.

6.6.2.1 Effect of Distance on External Radiation Risk Estimates. The sites containing
radionuclide contamination were examined for on-site risk from external radiation exposure. However, external radiation exposure from gamma emitting radionuclides may extend outward from a site boundary if the radiation is attenuated only by air, so external radiation dose may be additive if a receptor is in the proximity of several sites containing radionuclide soil contamination. As a result, it is necessary to evaluate the effect of distance on external radiation risk to determine if the dose received outside individual site boundaries is significant.

Since the WAG 1 release sites have a variety of shapes and may not have homogeneous radionuclide concentrations, exact calculation of radiation dose at specific points is not possible. However, by approximating the geometry of the sites, radiation doses at specific points adjacent to several sites is calculated.

Radiation dose and its associated risk decreases with distance from discrete sources. The rate of decrease with distance is dependent on the geometry of the source. External radiation dose from a horizontal plane source (e.g., an area of radionuclide contaminated soil) can be determined by assuming the total area of the plane is the source of external radiation.

To determine whether cumulative radiation doses could cause external radiation risks between release sites to be higher than radiation risks at each of the sites individually, the four release sites with the highest levels of contamination were identified and the exposure rate at a point in between sites was

calculated. Release sites TSF-06 (Area 8), TSF-06 (Area B), TSF-07, and TSF-10 are contaminated with radionuclides and are located fairly close to one another, so these sites were considered in the analysis.

Radionuclide soil concentrations at the test sites were reported in units of pCi/g of soil. To determine the radiation dose, these soil concentrations were converted to units of total activity, or Curies (Ci) of activity. The total activity for each radionuclide concentration was determined using the following formula.

$$Ci = C \times \rho \times A \times D \times CF_1 \times CF_2 \tag{6-24}$$

where

Ci	=	radionuclide activity (Ci)
C	=	radionuclide soil concentration (pCi/g)
ρ	=	soil density (g/cm^3)
A	=	release site area (m^2)
D	=	depth of contaminated soil (m)
CF_1	=	conversion from pCi to Ci (= 1E-12 Ci/pCi)
CF_2	=	conversion from m^3 to cm^3 (= 1E+06 cm^3/m^3)

Table 6-5 lists the calculated activity for each of the four test sites. In the radiation modeling performed for this evaluation, the total activity at each site was conservatively assumed to be uniformly distributed in 1 cm (2.5 in.) thick planes located at each site. Additionally, no credit was taken for shielding by soil, structures, or terrain.

Although not listed in the sampling concentrations, Ba-137m was included in the list of radionuclides. Ba-137m is the gamma-emitting daughter product of Cs-137. The half-life of Ba-137m is brief (2.5 minutes), so Ba-137m was assumed to be in secular equilibrium with its parent Cs-137. Since Cs-137 emit beta radiation (not an external hazard at these distances), it is necessary to include the effects of Ba-137m [which emits 0.662 MeV gamma radiation] to accurately determine the external gamma dose for all sites containing Cs-137. A similar situation exists for the isotopes Sr-90 and its daughter product, Y-90. The half-life of Sr-90 is 29.1 years, decaying by beta emission to Y-90. The Y-90 decay product, with a half life of 64 hours, transforms rapidly to the stable isotope Zr-90. Because the Y-90 decays at a greater rate than Sr-90, both isotopes quickly reach a state of equilibrium. However, since both Sr-90 and Y-90 decay by beta emission, there is no external radiation hazard from these isotopes.

The dose point used to determine the off-site cumulative external radiation dose is located 36 m (120 ft) north of TSF-10, and 110 m (360 ft) south of the railroad tracks. The point is centered near the sites with the highest estimated external radiation exposure risk as identified in Section 6.5. As a result, this location is considered to be the most conservative spot to determine the cumulative dose equivalent from all sites.

Table 6-5. Total activity (Ci) by site for depth range 0 to 4 ft (occupational scenario)

Radionuclide	Site			
	TSF-06(8)	TSF-06(B)	TSF-07	TSF-10
Am-241	—	—	3.08E–06	—
Ba-137m	1.46E–02	3.10E–05	7.48E–03	1.24E–04
Co-60	1.58E–04	2.51E–02	8.09E–03	—
Cs-134	—	—	1.92E–06	—
Cs-137	1.46E–02	3.10E–05	7.48E–03	1.24E–04
H-3	—	—	1.76E–04	—
Np-237	6.73E–08	—	—	—
Pu-238	9.13E–08	—	—	—
Ra-226	—	—	3.44E–04	—
Sr-90	4.61E–04	—	—	—
Th234	—	—	7.48E–03	—

In addition to estimating the cumulative dose rate at the exposure point, each dose rate immediately adjacent to the site is also determined (i.e., distance from each test site equals zero). These individual dose rates are included to illustrate the effect of distance on external radiation risk estimates.

The output from this model was used to construct Table 6-6, which presents the area of the site, the distance of the dose point from each site, the radiation flux and dose equivalent rate. The radiation flux is included to show the small amount of gamma radiation that will pass through the dose point. With so little radiation present at the evaluated dose point, the dose rate from each site is small, ranging in order from 2.0E–06 to 1.8E–04 millirem per hour (mrem/hr).

At the dose point, the cumulative occupational receptor dose rate from all of the test sites is calculated to be 2.27E–04 mrem/hr. Assuming an 8-hour work-day and a 250 day work-year, the annual dose rate for a worker standing at the dose point is 4.54E–01 mrem/yr.

Radiation doses are limited by DOE. DOE Order 5480.11, "Radiation Protection for Occupational Workers," limits radiation exposure of workers to 5 rem/year. DOE Order 5400.5, "Radiation Protection of the Public and the Environment," limits radiation exposures to members of the general public to 100 mrem/year. The calculated dose rate between the four test sites is approximately four orders of magnitude less than the 5 rem/year limit, and a factor of approximately 200 less than the 100 mrem/year limit

Table 6-6. Cumulative occupational dose rate (mrem/hr) for external gamma radiation.

OU Site Code	Area of Site (ft^2)	Distance (ft)	Flux (γ/cm^2 - sec)	Dose Rate (mrem/hr)
TSF-06(8)	13,625	681	1.50E–02	1.58E–05
		0	4.42E+00	4.61E–03
TSF-06(B)	160,312	620	9.44E–02	1.82E–04
		0	9.32E–01	1.98E–03
TSF-07	105,400	800	1.56E–02	2.72E–05
		0	4.95E–01	8.10E–04
TSF-10	28,206	288	1.98E–03	2.03E–06
		0	5.45E–03	5.57E–06
			Cumulative dose rate at dose point:	2.27E–04

6.6.3 Toxicity Assessment

Several important measures of toxicity are needed to conduct an assessment of risk to human health. RfDs are applied to the oral and inhalation exposure to evaluate noncarcinogenic and developmental effects, and SFs are applied to the oral and inhalation exposures to carcinogens. RfDs are derived from NOAELs or LOAELs and the application of uncertainty factors (UFs) and modifying factors (MFs). UFs are used to account for the variation in sensitivity of human subpopulations and the uncertainty inherent in extrapolation of the results of animal studies to humans, while MFs account for additional uncertainties in the studies used to derive the NOAEL or LOAEL. Uncertainty associated with SFs is accounted for by an assigned weight-of-evidence rating that reflects the likelihood that the toxicant is a human carcinogen. Weight-of-evidence classifications are tabulated and included in Table B-61, while a discussion of the UFs and MFs used to derive RfDs are presented in Section 6.4.

6.6.4 Risk Characterization

The last step in the risk assessment is risk characterization. As discussed in Section 6.5, risk characterization is the process of integrating the results of the exposure and toxicity assessments. The uncertainties defined throughout the analysis process are combined and presented as part of the risk characterization to provide an understanding of the overall uncertainty in the estimate of risk. Table 6-7 presents this qualitative assessment of uncertainty. See Section 8 for a summary of WAG 1 risks.

6.6.5 Assessment of Co-Located Facilities

Over the past 42 years, TAN has provided facilities, utilities, and support capabilities for government and private agencies to conduct experiments associated with the development, testing, and analysis utilized in aircraft nuclear, nuclear and reactor applications and military component fabrication. To support these missions, over 89 buildings and structures have been constructed. Through time facilities have been modified to fit TAN's changing mission. Currently, there are three major active programs located at TAN. These programs are the SMC project, Nuclear Operations supported by TAN Operations,

Table 6-7. BRA human health assessment uncertainty factors.

Uncertainty factor	Effect of uncertainty	Comment
Source term assumptions	May overestimate risk	All contaminants are assumed to be completely available for transportation away from the source zone. In reality, some contaminants may be chemically or physically bound to the source zone and unavailable for transport.
Natural infiltration rate	May overestimate risk	A conservative value of 10 cm/year was used for this parameter.
Moisture content	May overestimate or underestimate risk	Soil moisture contents vary seasonally in the upper vadose zone and may be subject to measurement error.
Water table fluctuations	May slightly overestimate or underestimate risk	The average value used is expected to be representative of the depth over the 30-year exposure period.
Mass of contaminants in soils is estimated by assuming a uniform contamination concentration in the source zone.	May overestimate or underestimate risk	There is a possibility that most of the mass of a given contaminant at a given site may exist in a hotspot that was not detected by sampling. If this condition existed, the mass of the contaminant used in the analysis might be underestimated. However, 95% UCLs or maximum detected contamination were used for all mass calculations, and these concentrations are assumed to exist at every point in each waste site; therefore, the mass of contaminants used in the analysis is probably overestimated.
Plug flow assumption in groundwater transport	Could overestimate or underestimate risk	Plug flow models are conservative with respect to concentrations because dispersion is neglected, and mass fluxes from the source to the aquifer differ only by the time delay in the unsaturated zone (the magnitude of the flux remains unchanged). For nonradiological contaminants, the plug flow assumption is conservative because dispersion is not allowed to dilute the contaminant groundwater concentrations. For radionuclides, the plug flow assumption may or may not be conservative. Based on actual travel time, the radionuclide groundwater concentrations could be overestimated or underestimated because a longer travel time allows for more decay. If the concentration decrease because the travel time delay is larger than the neglected dilution from dispersion, the model will not be conservative.
No migration of contaminants from the soil source prior to 1994	Could overestimate or underestimate risk	The effect of not modeling contaminant migration from the soil before 1994 is dependent on the contaminant half-life, radioactive in growth, and mobility characteristics.
Contaminant source terms assumed to be lognormally distributed	Could overestimate risk	If sampling data at a given site fits a normal distribution rather than a lognormal distribution, the 95% UCL of the near concentrations calculated for the site could be as much as 50% too high. EPA from Superfund sites are lognormally distributed (EPA 1992).
Chemical form assumptions	Could overestimate or underestimate risk	In general, the methods and inputs used in contaminant migration calculations, including assumptions made about the chemical forms of contaminants were chosen to err on the protective side. All contaminant concentration and mass are assumed available for transport. This assumption results in a probable overestimate of risk.
Exposure scenario assumptions	May overestimate risk	The likelihood of future scenarios has been qualitatively evaluated as follows: resident - improbable industrial - credible. The likelihood of future onsite residential development is small. If future residential use of this site does not occur, then the risk estimates calculated for future onsite residents are likely to overestimate the true risk associated with future use of this site.

6-70

Table 6-7. (continued).

Uncertainty factor	Effect of uncertainty	Comment
Exposure parameter assumptions	May overestimate risk	Assumptions regarding media intake, population characteristics, and exposure patterns may not characterize actual exposures.
Receptor locations	May overestimate risk	Groundwater ingestion risks are calculated for a point at the downgradient edge of an equivalent rectangular area. The groundwater risk at this point is assumed to be the risk from groundwater ingestion at every point within the TAN boundaries. Changing the receptor location will affect only the risks calculated for the groundwater pathway because all other risks are site-specific or assumed constant at every point within the TAN boundaries.
For the groundwater pathway analysis, all contaminants are assumed to be homogeneously distributed in a large mass of soil.	May overestimate or underestimate risk	The total mass of each COPC is assumed to be homogeneously distributed in the soil volume beneath Test Reactor Area (TRA). This assumption tends to maximize the estimated groundwater concentrations produced by the contaminant inventories because homogeneously distributed contaminants would not have to travel far to reach a groundwater well drilled anywhere within the TAN boundary. However, groundwater concentrations may be underestimated for a large mass of contamination located in a small area with a groundwater well drilled directly downgradient.
The entire inventory of each contaminant is assumed to be available for transport along each pathway	May overestimate risk	Only a portion of each contaminant's inventory is actually transported by each pathway.
Exposure duration	May overestimated	The assumption that an individual will work or reside at TAN for 25 or 30 years is conservative. Short-term exposures involve comparison to subchronic toxicity values, which are generally less restrictive than chronic values.
Noncontaminant-specific constants (not dependent on contaminant properties)	May overestimate risk	Conservative or upper limit values were used for all parameters incorporated into intake calculations.
Exclusion of some hypothetical pathways from the exposure scenarios	May underestimate risk	Exposure pathways are considered for each scenario and eliminated only if the pathway is either incomplete or negligible compared to other evaluated pathways.
Poorly defined dermal absorption factors (ABS) values for most WAG 1 contaminants	May underestimate risk	A lack of ABS values for most WAG 1 contaminants may mean that dermal absorption risks are higher than expected. The possibility of unacceptable dermal absorption from soil risks being produced by WAG 1 contaminants is considered to be unlikely.
Model does not consider biotic decay	May overestimate risk	Biotic decay would tend to reduce contamination over time.
Occupational intake value for inhalation is conservative	Slightly overestimates risk	Standard exposure factors for inhalation have the same value for occupational as for residential scenarios although occupational workers would not be onsite all day.
Use of cancer SFs	May overestimate risk	Nonradionuclide SFs are associated with upper 95th percentile confidence limits and radionuclide SFs are central estimates of cancer incidence per unit intake. They are considered unlikely to underestimate true risk.
Toxicity values are derived primarily from animal studies	May overestimate or underestimate risk	Extrapolation from animal to humans may induce error caused by differences in absorption, pharmacokinetics, target organs, enzymes, and population variability.
Toxicity values are derived primarily from high doses; most exposures are at low doses	May overestimate or underestimate risk	Assumes linearity at low doses. Tends to have conservative exposure assumptions.

Table 6-7. (continued).

Uncertainty factor	Effect of uncertainty	Comment
Toxicity values and classification of carcinogens	May overestimate or underestimate risk	Not all values represent the same degree of certainty. All are subject to change as new evidence becomes available.
Lack of SFs	May underestimate risk	COPCs without SFs, may or may not be carcinogenic through the oral pathway.
Lack of RfDs	May underestimate risk	COPCs without RfDs may or may not have noncarcinogenic adverse effects.
Risk/HQs are combined across pathways	May overestimate risk	Not all of the COPC inventory will be available for exposure through all applicable exposure pathways.

and Research and Engineering Laboratory Activities . The SMC, a Department of Army facility, was specifically omitted from the FFA/CO. The remaining facilities and structures are used support these TAN programs or other INEEL missions.

In view of the fact that past and present activities associated with TAN facilities and structures are proximal or "co-located" to WAG 1 sites identified in the FFA/CO, an analysis was performed to address their potential for causing current risk to be under estimated. This analysis was performed in addition to previous investigations that have been conducted at WAG 1 and it should be noted that the majority of the structures identified as having the potential for past or future releases were included in these previous investigations. The complete co-located facilities/structures analysis is contained in Appendix D to this report.

This analysis includes a review of past and present operational activities at TAN and associated facilities and structures, and management control procedures to prevent and mitigate releases. All operational facilities/structures, facilities/structures no longer being utilized for their original mission, and facilities in standby, or abandoned mode are included in this analysis. In support of the analysis, a set of screening and retention criteria for building/structures and related activities was developed.

An integral part of the analysis is the review of management control procedures utilized to mitigate potential releases to the environment at TAN. These controls include Safety Analysis Reviews for nuclear facilities, Resource Conservation and Recovery Act (RCRA) Contingency Plans, Spill Avoidance and Response Plans, Emergency Plan Implementing Procedures, and Nuclear Materials Inspection and Storage Procedures. These procedures are designed to specifically address potential releases to the environment at TAN and appropriate reporting and mitigation measures to be implemented in such an event. In support of these management control procedures are standard operating procedures that cover operational aspects of activities at TAN. These procedures are designed to eliminate or minimize the risk of off normal events. In addition to TAN specific management control procedures, the site contractor has Site-wide program requirements. These program requirements include physical hazards, asbestos control, and toxic substance control.

6.6.5.1 Summary of Co-Located Facility Risk at Retained Sites.
Based on the analysis performed of co-located facilities and activities and management controls to prevent releases to the environment, only the TAN Hot Shop facility, the Radioactive Parts Security Storage Area (RPSSA) building outside asphalt pads, which is a Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) site, and the two Radioactive Liquid Waste Treatment and Transfer/Storage buildings are identified to have the potential to impact comprehensive risk at WAG 1. The analysis did not identify any structures or facilities that posed an imminent threat of release; however, the retention of these areas is based primarily on remote accident scenarios or on the fact past releases have been documented at these or similar sites. Data concerning past releases from these sites and investigations conducted to characterize them are contained in Section 3 of this report. Documented releases have occurred to the soil beneath the pads surrounding the outside of the RPSSA buildings. The Hot Shop facility and the RPSSA storage buildings are part of the active operations at TAN. The retained structures are summarized below.

6.6.5.2 TAN Hot Shop Facility.
The Hot Shop facility, for the purposes of this analysis, is defined as consisting of the TAN-607 Hot Shop, the Hot Shop Pool and support areas, are parts of building TAN-607. The primary area of concern is the Hot Shop Pool. This facility has not experienced past releases to the environment, but has the potential during a catastrophic earthquake to develop a leak and lose part or all of the water, which acts as shielding to the nuclear materials stored in the pool. A more complete description of the area is contained in Appendix D.

6.6.5.3 TAN RPSSA Building Pads. The RPSSA buildings (TAN-647 and TAN 648) are two large storage areas that have been screened from being a potential threat of causing a release to the environment during the analysis of co-located facilities. The asphalt pads, however, that surround the buildings cover and fix radioactive contaminated soil, causing this site to be retained for further evaluation. Data concerning this contamination and investigations conducted to characterize the area are contained in Section 3 of this report. A more complete description of the area is contained in Appendix D.

6.6.5.4 Radioactive Liquid Waste Treatment and Transfer/Storage Buildings. The Radioactive Liquid Waste Treatment building (TAN-616) and the Radioactive Liquid Waste Transfer and Storage building (TAN-666) are part of the system that treated, stored and transferred radioactive contaminated liquid waste that was generated from facilities such as the TAN Hot Shop, Hot Cell, and the Hot Cell Annex. These facilities are connected by the TSF Intermediate Waste disposal system discussed in Section 3 of this report. Neither building is currently in use as radioactive liquids are no longer generated in these facilities. A more complete description of these buildings are contained in Appendix D.

6.6.5.5 Summary and Conclusions. Based on the analysis performed of co-located facilities and activities and management controls to prevent potential past and future releases to the environment, only the TAN Hot Shop facility, the RPSSA building outside asphalt pads, which is a CERCLA site, and the two Radioactive Liquid Waste Treatment and Transfer/Storage buildings were identified to have the potential to impact comprehensive risk at the TAN. The analysis included both the active and inactive systems at TAN. The analysis did not identify any structures or facilities that posed an imminent threat of release; however, the retention of these areas is based primarily on remote accident scenarios or on the fact past releases have been documented at these or similar sites. The Hot Shop facility, for the purposes of this analysis, is defined as consisting of the TAN-607 Hot Shop, the Hot Shop Pool and support areas. The Hot Shop area would require a Zone 3 earthquake that could cause the pool to crack causing loss of shielding (water) before this area would impact the environment. The settlement agreement between the State of Idaho, DOE, and Department of the Navy (Batt Agreement) requires the TMI Fuel to be removed from the pool by June 1, 2001. It is planned that all other materials stored in the pool will be removed at that time also. The contamination outside of the RPSSA buildings is covered by asphalt and fixed in place. The Radioactive Liquid Waste Treatment and Transfer/Storage buildings are currently unused, with the treatment building scheduled for removal in FY 1998. All of the sites represent structures that are part of active operations at TAN which are covered under appropriate management control procedures. The potential for these retained sites to impact current risk estimates is very remote.

6.6.6 Sensitivity Analysis

A sensitivity analysis was performed to assess potential difference in risk estimates given changes to the exposure assumptions used in the human health assessment for OU 1-10. The objective of the analysis was to illustrate the magnitude of risk reduction achieved by changing select assumptions and the corresponding numerical values used in the calculations. To meet this objective, three baseline assumptions for a hypothetical future resident were modified:

1. Keeping the exposure point concentrations constant, the exposure parameters were modified to reflect probable and more realistic future resident exposures

2. Keeping the exposure point concentrations constant, the exposure parameters were modified to reflect conditions for a modified (i.e., not full time) resident

3. Keeping the exposure assessment assumptions constant, alter the exposure point concentrations to reflect average rather upper bound conditions (this scenario is currently still under development).

The following sections discuss the assumptions used in the sensitivity analysis and the observed impacts to the risk estimates.

6.6.6.1 *Exposure Parameter Sensitivity.* Table 6-8 illustrates the exposure assessment parameters that were altered for the future resident and the modified resident in the sensitivity analysis. Generally, the parameters used for the future resident (averaging time, exposure duration, exposure frequency, exposure time and ingestion rate) reflect more typical residential exposures than the parameter values used in the BRA base case analysis (see Section 6.5). The only parameters altered for the modified resident were exposure frequency and exposure time. Parameters not shown here are assumed to be consistent with those used in the BRA base case analysis.

Applying the more realistic exposure parameter assumptions to the future resident resulted in a reduction in the total risk estimated by 50 to 60% (i.e., a risk reduction factor of 0.5 to 0.4). The modified resident assumptions resulted in a reduction in the total risk estimated by 60 to 99 % (i.e., a risk reduction factor of 0.4 to 0.01). For example the total risk estimate for TSF-36 under the baseline conditions is 5E-05. Apply a risk reduction factor of 0.4 to this estimate would result in a risk of 2E-05. For the two cases considered (i.e., the future resident and the modified resident), modifying the resident assumptions from a full-time resident to a part-time resident resulted in a greater reduction in risk than assigning more realistic exposure parameters.

6.6.6.2 *Exposure Point Concentration Sensitivity.* Altering the exposure point concentrations to reflect averaged rather than upper bound conditions resulted in a reduction in the total risk estimated by 33 to 86% (i.e., a risk reduction factor of 0.67 to 0.14). For example the total risk estimate for TSF-07 under baseline conditions is 7E-04. Applying a risk reduction factor of 0.14 to this estimate would result in a risk of 6E-04.

Table 6-8. Sensitivity analysis exposure parameter values and sources.

Exposure Parameter	Modified Parameter Value	Rationale/Source
Averaging Time (Noncarcinogenic)		
Future resident	7,300 days	Adult, noncarcinogenic, 20 years × 365 days.
Exposure duration		
Future resident	20 years	Exposure factors handbook indicates 77% of residents move prior to 20 years.
Exposure frequency		
Future resident (soil exposure)	305 days/year	Assumes no exposure to soil for 60 days/year during periods of snowpack and/or frozen ground.
Modified resident	150 days/year	Assumed exposure frequency
Exposure time		
Future resident	12 hours/day	Exposure factors handbook indicates an average of 730 minutes/day are spent on non-residential activities
Modified resident	0.25 hours/day	Assumed exposure time
Ingestion rate		
Future residential (soil)	60.5 mg/day	Exposure factors handbook provides mean ingestion rate
Future residential (water)	1.36 L/day	Exposure factors handbook provides mean ingestion rate

6.7 References

Amdur, M. O., T. Doull, and C. D. Klassen, 1991, *Casarett and Doull's Toxicology, The Basic Science of Poisons*, 4th ed., Pergaman Press.

Andelman, J. B., 1990, *Total Exposure to Volatile Organic Compounds in Potable Water*, in: *Significance and Treatment of Volatile Organic Compounds in Water Supplies*, Lewis Publishers, Inc., Chelsea, Michigan.

ATSDR (Agency for Toxic Substance Disease Registry), 1989, *Toxicological Profile for Plutonium*.

ATSDR, 1990, *Toxicological Profile for Uranium*.

Baes, C. F. III, and R. D. Sharp, 1993, *A Proposal for Estimation of Soil Leaching and Leaching Constants for Use in Assessment Models*, Journal of Environmental Quality, 12, 1, pp. 17–28.

BEIR IV, 1988, *Health Effects of Radon and Other Internally Deposited Alpha-Emitters,* Committee on Biological Effects of Ionizing Radiations, National Research Council, National Academy Press, Washington, D.C.

Cornfield, J., 1977, *Carcinogenic Risk Assessment,* Science, 198:693–699.

Crump, K. S., H. A. Guess, and K. L. Deal, 1977, *Confidence Intervals and Test of Hypotheses Concerning Dose Response Relations Inferred from Animal Carcinogenicity Data, Biometrics,* 333:437–451.

DOE-ID (U.S. Department of Energy, Idaho Operations Office), 1994, *Track 2 Sites: Guidance for Assessing Low Probability Hazard Sites at the INEL,* DOE/ID-10389, Rev. 6, January.

DOE-ID, 1996, *Work Plan for Waste Area Group 1 Operable Unit 1-10 Comprehensive Remedial Investigation/Feasibility Study,* DOE/ID-10527, March.

DOE-ID, 1996, *Comprehensive Facility and Land Use Plan,* DOE/ID-10514, March.

EPA (Environmental Protection Agency), 1986, *Guidelines for Carcinogen Risk Assessment,* Federal Register, 51:33992–34003.

EPA, 1989a, *Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual (Part A),* EPA/540/1/1-89/002, December.

EPA, 1989b, "Mouse Oral Subchronic Study with Acenaphthene," Study conducted by Hazelton Laboratories, Inc., for the Office of Solid Waste, Washington, D.C.

EPA, 1991, *Supplemental Risk Assessment Guidance for Superfund,* EPA Region 10, August.

EPA, 1992a, *Guidance for Data Usability in Risk Assessment (Parts A and B),* EPA-9285.7A and B, April and May.

EPA, 1992b, *Supplemental Guidance to RAGS: Calculating the Concentration Term,* Office of Solid waste and Emergency Response, Washington, D.C., EPA Publication 9285.7-081, May.

EPA, 1993, *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons,* Environmental Criteria and Assessment Office.

EPA, 1994, Health Effects Assessment Summary Tables, EPA 540-R-94-020, November.

EPA, 1995, *Assessing Dermal Exposure from Soil,* EPA/903-K-95-003, December.

Ghetti, G., and Mariana, 1956, *Eye Changes Due to Naphthalene, Archives of Ophthalmology,* 41:572–582.

Hardin, B. D., et al., 1981, *Testing of Selected Workplace Chemicals for Teratogenic Potential,* Scandinavian Journal of Work and Environmental Health, 7:66–25.

Hoff, D. L., R. G. Mitchell, R. Moore, and L. Bingham, 1993, *The Idaho National Engineering Laboratory Site Environmental Report for Calendar Year 1992*, DOE/ID-12082, June.

Horne, R. A., 1978, *The Chemistry of our Environment*, John Wiley and Sons, New York, NY.

IAEA (International Atomic Energy Agency), 1994, *Handbook of Parameter Values for the Prediction of Radionuclide Transfer in Temperate Environments*, Technical Reports Series No. 364.

Kirchman, R., et al., 1993, *Terrestrial Pathways, Radioecology After Chernobyl*, E. E. Warner and R. M. Hamson (eds), John Wiley and Sons, New York, NY.

Lewis et al., 1996, *Work Plan for Waste Area Group Operable Unit 1-10 Comprehensive Remedial Investigation/Feasibility Study*, DOE/ID-10527, March.

LMITCO (Lockheed Martin Idaho Technologies Company), 1995, *Guidance Protocol for the Performance of Cumulative Risk Assessments at the INEL*, INEL-95/131, Rev. 0, May.

LMITCO, 1996, *White Paper on the Food Crop Ingestion Exposure Pathway*, Rev. 2, INEL-95/0275, May.

Mahavei, D., H. Bank, and R. Harvey, 1977, *Morphology of a Naphthalene Induced Bronchiolar Lesion*, American Journal of Pathology, 86:559–571.

Maheras, S. J., et al., 1994, *Radioactive Waste Management Complex Low-Level Waste Radiological Performance Assessment*, EGG-WM-8773, May.

Mantel, N., et al., 1971, *An Improved "Mantel-Brown" Procedure to Safety Testing of Carcinogens*, Cancer Res., 35:865–872.

Manzo, L., et al., 1992, *Metabolic Studies as a Basis for the Interpretation of Metal Toxicity*, Toxicity Letters, 64165:677–686.

NAS (National Academy of Sciences), 1980, "The Effects on Populations of Exposure to Low Levels of Ionizing Radiation (BEIR III)," National Academy Press, Washington, DC.

NRC (Nuclear Regulatory Commission), 1993, "Radiological Assessment: A Textbook on Environmental Dose Analysis," J. E. Till and H. R. Meyers, eds, NUREG/CR-3332, ORNL-5968, September.

Rood, A. S., 1994, *GWSCREEN: A Semi-Analytical Model for the Assessment of the Groundwater Pathway from Surface or Buried Contamination: Version 2.0, Rev. 2.0*, Theory and User's Manual, EGG-GEO-10797, June.

Rood, S. M., G. A. Harris, and G. J. White, 1995, *Background Dose Equivalent Rates and Surficial Soil Metal and Radionuclide Concentrations for the Idaho National Engineering Laboratory*, INEL-94/0250, Rev. 0, February.

Scheuhammer, A. M., 1987, "The Chronic Toxicity of Aluminum, Cadmium, Mercury, and Lead in Birds: A Review," *Environmental Pollution*, 46(4):263–296.

Sherman, 1974, *Long-term feeding studies in rats and dogs with dichlorodifluoromethane (Freon 12 Food Freezant)*, Haskell Laboratory for Toxicology and Industrial Medicine Report No. 24–74.

Shopp, G. M., et al., 1984, *Naphthalene Toxicity CD-1 Mice*, General Toxicology and Immunotoxicology, Fundamentals of Applied Toxicology id, 4:406–419.

VanHorn, R. L., N. L. Hampton, and R. C. Morris, 1995, *Guidance Manual for Conducting Screening Level Ecological Risk Assessments at the INEL*, INEL-95/0190, April.

Wren, C. D., 1986, "A Review of Metal Accumulations and Toxicity in Wild Mammals," I., Mercury, *Environmental Research*, 40(1):210–44.

Zvelzer, W. W., and L. Apt, 1949, *Acute Hemolytic Anemia Due to Naphthalene Poisoning*, JAMA, 141:185–190.